CYTODIAGNOSIS OF ORAL LESION WITH HISTOPATHOLOGICAL CORRELATION

THESIS
FOR
DOCTOR OF MEDICINE
(PATHOLOGY)

BUNDELKHAND UNIVERSITY
JHANSI (U P)

1990

Surender Katyal

C E R T I F I C A T E

This is to certify that the work entitled "CYTODIAGNOSIS OF ORAL LESIONS WITH HISTOPATHOLOGICAL CORRELATION" being submitted for M.D.(PATHOLOGY) has been carried out by DR. SURENDER KATYAL himself in this department.

He has put in the necessary stay in the department as required by the regulation of Bundelkhand University, Jhansi.

(R.K.GUPTA)
M.D., MNAMS,
Professor and Head
Department of Pathology,
M.L.B. Medical College,
Jhansi

Dated : 8/1/90

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This is to certify that the work entitled "CYTODIAGNOSIS OF ORAL LESIONS WITH HISTOPATHOLOGICAL CORRELATION" being submitted for M.D. (PATHOLOGY) has been carried out by DR. SURENDER KATYAL under my guidance and supervision and, in the Department of Pathology. His observations and results have been checked and verified by me from time to time.

This work fulfils the basic ordinances governing the submission of thesis laid down by Bundelkhand University, Jhansi.

V.K.S

(V.K. SHARMA)
M.D., D.C.P.,
Lecturer,
Department of Pathology,
M.L.B. Medical College,
Jhansi

Dated :

GUIDE

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(D.C. GOVIL)
M.D., D.V.D.,
Reader and Head
Department of Skin & V.D.,
M.L.B. Medical College,
Jhansi

CO-GUIDE

(R.P. SRIVASTAVA)
B.Sc., BDS, MDS (Paric
Professor and Head
Department of Dental Surgery
M.L.B. Medical College,
Jhansi

CO-GUIDE

(J.P. PUROHIT)
M.S.,
Lecturer in E.N.T.,
M.L.B. Medical College,
Jhansi

Dated :

CO-GUIDE

A C K N O W L E D G E M E N T

Words are sometimes hard to find when one tries to say thanks for something, so priceless as loving criticism, considerate helpfulness and valuable guidance. Too much gratitude and sincerity repel and too little leave one wanting, though facts must be evidently acknowledged and honest thankfulness must be unequivocally stated. This is what I have humbly attempted to do here.

The affectionate nature and heartening words of Prof. R.K. Gupta, M.D., MNAMS, Professor and Head, Department of Pathology, M.L.B. Medical College, Jhansi, constantly provided me the confidence and enthusiasm, so essentially vital, to such a project.

It was my proud privilage to have been associated with and remain under the constant vigilance of Dr. V.K. Sharma, M.D.,D.C.P., Lecturer, Department of Pathology, M.L.B. Medical College, Jhansi. This study is a reflection of discerning criticism and seasoned rationale of him. His uncompromising standards and masterly guidance set the trend and pace for this work.

In no less degree I owe my sincerest thanks to my Co-guides Dr. R.P. Srivastava, B.Sc., BDS, MDS (Paris), Professor and Head, Department of Dental Surgery, Dr. D.C. Gavil, M.D., D.V.D., Reader and Head, Department of Skin & V.D. and Dr. J.P. Purohit, M.S., Lecturer in E.N.T. Department, M.L.B. Medical College, Jhansi. They have been too kind to help me even at their personal inconvenience of every stage of their work.

I am also grateful to Dr. Ratna, M.D., Lecturer in Pathology and Dr. Rajeev Sinha, M.S., Lecturer in Surgery, M.L.B. Medical College, Jhansi for their kind help and guidance.

I was really fortunate to have the enjoyment as well as benefit of working with a superb team of colleagues, each one of them spared no efforts to be of utmost use and help to me, inspite of various engagements and duties of theirown.

It gives me special pleasure to acknowledge the help extended and moral support provided by my wife Dr. Mridula, and my parents during my hours of desperation due to ever arising problems and time consuming process.

And in last but not the least I will in my duty if I do not offer my sincere thanks to Mr. Kanhaiya Lal for the pains taken by him in bringing out such a neat type script.

Surender Katyal.

Dated : 5.2.90. (SURENDER KATYAL)

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I N T R O D U C T I O N

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Multitier investigations in oncopathology is increasing day by day as clinician and cytopathologist are encountering varied picture in cancer pathology.

Advanced countries have assimilated sophistication heavily in oncopathology (like tumour marker, tumour antibodies etc). But we are still in search of an efficient screening method by which the cytopathologist can point accurately towards the nature of disease within a few hours.

Oral cancer is a significant health problem accounting for approximately 5 per cent of all malignant tumours involving the body (Shklar, Meyer, Cataldo and Taylor, 1968). Oral cancer though not so common in the western countries, is quite prevalent in this part of the world. In 1902, Niblock first reported the high incidence of oral cancer particularly that of buccal mucosa in India. Bentall in 1908, reported the frequency of oral cancer in this part of the globe to be 38% among all cases of cancer. Since then, innumerable studies by authors like Kini and Rao (1937), Khanolkar et al (1950), Baruah (1964), Paymaster (1964), Wahi et al (1965), Chitkara et al (1966), Reddy et al (1967) and many

others uniformly indicated the preponderance of oral cancers throughout the country. International Union against Cancer (U.I.C.C.) 1970, in its publication reported higher incidence of cancer of mouth, tongue and pharynx in India in relation to other countries.

Oral malignancies and pre-malignant or pre-cancerous lesions like leukoplakia, melanoplakia, erythroplakia, sub-mucous-fibrosis, lichen planus, stomatitisnicotina palati, non-healing ulcers etc., are very common in our country, mainly due to heavy consumption of tobacco in varied form and the contributory factors like alcohol, poor oral hygiene, syphilis etc. Thus a reliable method for early diagnosis of these diseases is extremely desirable in order that correct treatment can be instituted at a stage when the chances for cure are infinitely greater. "Cytological study" is hoped to achieve that goal. In the past decade or so the technique of cytodiagnosis has been used in evaluating oral disease. Umiker et al (1960), Sandler (1964), Hayes et al (1969), and King (1971) are among some of those who have generally supported the thesis that oral exfoliative cytology is a reliable and sensitive diagnostic tool.

Numerous reports (Allegra, 1973; Falaom 1972; Hayes 1969; Sandler 1964) substantiate that the use of oral cytology has accelerated biopsy of lesions which clinically did not appear to be oral cancers and has led to early diagnosis of cancer which would otherwise have remained temporarily unsuspected.

Cytological study is simple, bloodless, painless, rapid and easily acceptable to patient and hence, can be repeated number of times as compared to biopsy which is tedious and not acceptable to everyone. The introduction of fluoresacent microscopy, phase contrast microscopy and automatic cytoscanner has brought newer advances to this diagnostic field. However, even in absence of those sophisticated instruments, the technique holds its ground as a rapid diagnostic procedure. Hence its value in the diagnosis of oral lesions needs further study.

"AIMS OF THE STUDY"

The present study was conducted with a view :-

1. To assess the incidence of pre-cancerous and cancerous lesion of oral cavity in this part of country.

2. To fascilitate the confirmation of the clinical diagnosis of oral lesions.

3. To correlate cytopathological findings with histopathological observation and to assess the efficacy of cytology as an early diagnostic tool.

MATERIAL AND METHOD

MATERIAL AND METHOD

For the present study "CYTODIAGNOSIS OF ORAL LESIONS WITH HISTOPATHOLOGICAL CORRELATION" the material from the oral lesions was collected from the patients attending the various Out Patient Departments as well as from admitted patients in the wards of Maharani Laxmi Bai Medical College, Jhansi.

The relevant clinical data from all the patients was recorded along with the general examination, systemic examination, local findings, personal and family history on a preset proforma for further analysis.

COLLECTION OF MATERIAL FOR STUDY :

From each patient material was collected for cytopathological and histopathological study.

(1) Material for cytopathological study :

For cytopathological examination smears from oral lesions were prepared by scrap method using wooden spatula and preserved in a suitable fixative.

(2) Material for histopathological examination :

Biopsies were taken from suspected oral lesions in suitable cases for the confirmation and correlation with cytopathological examination.

kept at hand in a glass jar, without allowing it to dry. Atleast two smears in two slides from each lesion is thus collected scraping an wide area of the lesion. The slides are then marked suitably for future identification and reference.

HISTOPATHOLOGY :

In the same sitting, biopsy of the patient (if agreed for it) is taken from the local lesion and the tissue is kept in small formaline containing vial for preservation with a label mentioning serial No., date, name, age, sex, diagnosis, site of the lesion etc. The smears slides and biopsies are brought to pathology department of Maharani Laxmi Bai Medical College, Jhansi for cytological and histopathological examinations respectively.

FIXATIVE FOR ORAL SMEARS :

(1) Smears were taken from the local lesion and were kept in mixture of ether and alcohol (both in equal quantity) for 24 hours for fixation and preservation. During placing of slides in the jar with fixative, care was taken to keep the slides without touching one another and slides were put into fixative while still moist.

(2) Cytofix.

STAINING OF THE SMEARS :

Two stains were used for the study.

(i) Papanicolaou stain (1942) which is widely used for cytological examination and

(ii) Haematoxylin and Eosin stain, which is usually used for histopathological examination.

PAPANICOLAOU STAINING METHOD (1942) :

The different steps of the technique are as follows :-

(1) The fixed smears are at first carried through a process of hydration by dipping them for about 30 second in each, descending grades of alcohol (80%, 70% and 50%) to water.

(2) The hydrated smears are then dipped in Harris's Alum Haematoxylin and kept there for 5 minutes.

(3) The slides are then rinsed in tap water to wash out the excess stains.

(4) Differentiated in acid alcohol.

(5) The smears are then washed in running tap water for atleast 5 minutes.

(6) The smears are then completely dehydrated again by carrying them through 50%, 60%, 70%, 80%,90% and two changes of absolute alcohol for about 30 seconds in each.

(7) The completely dehydrated smears are put into the jar containing the stain, G.G. 6 and kept these for about 2 minutes and then rinsed again in two changes of absolute alcohol.

(8) The rinsed smears are then stained with the stain E.A.-50 by keeping them dipped into the jar containing it for a period of approximately 3 to 5 minutes.

(9) The smears thus stained are rinsed in two changes of absolute alcohol once more.

(10) The rinsed stained smear finally drained and cleared in two changes of xylol.

(11) Mount in D.P.X.

HAEMATOXYLIN AND EOSIN STAINING :

For histopathological study paraffin sections were prepared and stained by routine haematoxylin eosin staining technique as follows :-

(1) Bring section to water.

(2) Stained sections in haematoxylin for 10 minutes.

(3) Differentiated in acid alcohol until red (concentrated hydrochloric acid 1c.c., 70% alcohol 99 c.c.).

(4) Washed thoroughly in tap water.

(5) Counter stained 1 minute in 1% eosin.

(6) Rinsed in water.

(7) Dehydraded with 2 changes each of 95% and 100% alcohol.

(8) 100% + Xylene, then 2 changes of xylene.

(9) Mounted in D.P.X.

In suitable cases where biopsies were available, cytological findings were further confirmed and correlated with histopathological findings.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL REVIEW/BACKGROUND OF ORAL CYTOLOGY :

The interest of oral exfoliative cytology dates back to 1843 when Walshe first microscopically detected tumour particles in the sputum of a patient.

The first attempt at the cytologic diagnosis of pharyngeal cancer by oral smear was made over a century ago, in 1860, by Beale.

Hampelin in 1876 and Betechardt in 1895 contributed additional observation of malignant cells in the sputum of patients with carcinoma of the pharynx, lungs and bronchi.

The first modern application of oral cytology was that of Morrison and co-workers in 1949, who used the smear technique to diagnose nasopharyngeal lesions. They concluded that (i) the results were excellent,
(ii) the procedure was not a substitute for biopsy,
(iii) the technique was a valuable adjunct and positive results demands that the source be sought
(iv) a knowledge of normal cytology is essential
(v) strict attention must be paid to detail and
(vi) that cytologist must be experienced to evaluate the oral smear. These conclusions, certainly are still true today.

In 1943, Papanicolaou and Traut published their technique of staining the cells for proper and correct identification and study. Since the publication of Papanicolaou's staining technique, exfoliative cytology came to be recognized as a reliable diagnostic tool specially in identifying early cancers in different sites and effusions.

Montgomery (1951) studied the oral cytology of normal patient and emphasized the degree of variation that can be seen in the absence of disease.

Miller and Co-workers (1951) reported differences in the degree of cornification of oral mucosa depending on its anatomical location and Peters (1954) described the different cell types found in oral smears.

Several workers (Pomeranz and Stahl, 1953; Wahi and Gupta, 1954) applied this technique to known or clinically suspected oral carcinoma, to study its usefulness and to study different types of cells present in oral cytology.

Criterias for the interpretation of malignancy in oral smears have been listed by several investigators (Cawson, 1960; Hopp, E.S., Montgomery and Haam, 1951 ; Peters, H. and Rijsinghani, 1956).

Various methods were tried to collect a suitable specimen for study. Gladstone and Sidney (1950) used both gelfoam and cellulose sponze to collect specimen of representative cells. Montgomery (1951) used Woodson's No.2 metal plastic instrument and Pomermanz and Stahl (1951) used a wooden spatula to scrap the lesion and got satisfying results. Hopp (1958) however, found the tightly wound cotton applicator to be a handy as well as simple tool to collect specimen by firmly rubbing over the lesion and adjacent areas. Helsper, Sharp and Bullock(1963) presented the mouth wash technique to screen for intra oral carcinoma by which it was possible to get representative cells from all parts of the oral cavity. Scheman, Lumerman and Altchuler (1968) introduced 'Cytoaspirator' an instrument of their own design, for deep suction abrasion method of cytologic sampling. Malberger (1974) used aspiration biopsy to collect specimen from deeply seated orofacial masses. Camillert (1968) remarked exfoliative cytology to be an established method in the diagnosis of neoplastic and non-neoplastics lesions of the oral cavity. However, cent percent diagnostic accuracy has not been established by this method.

THE NORMAL ORAL MUCOSA

Knowledge of normal conditions is the prerequisite for diagnosis of pathological changes. The normal mucosa presents a moist, glistening surface, and is a rose

or greyish-pink colour due to the vascular bed in the connective tissue underlying the epithelium being well supplied with blood. The movability of the oral mucosa varies, and distinction is made between locolabile (lip, cheek, tongue, floor of mouth) and locostable (hard palate, gums) areas (Table - 1).

Table - 1 : Histological Structure of the Oral Mucosa

	Locolabile mucosa	Locostable mucosa
Stratified epithelium		
Basal layer	+	+
Prickle-cell layer	+	+
Granular layer	-	+/(+)
Horny layer	+	+/++
Orthokeratosis (Anucleate)	-	+
Parakeratosis (Nucleate)	+	-
Rete pegs/connective tissue papillae.	Shallow	deep
Lamina propria	Wide, loose	narrow, fibrous
Occurrence	lip, cheek, tongue, floor of mouth.	palate, gingiva

Histological examination shows the surface to be covered with a stratified squamous epithelium, with the sequence of layers similar to that seen in the epidermis of the skin. A basal cell layer (Stratum basale), spinous layer (stratum spinosum) and flattened horny layer (stratum corneum) may be distinguished. A granular layer (stratum granulosum) is found only in areas showing orthokeratotic keratinisation. However, as a rule, there is clearly less keratinisation than in the epidermis. Differentiation is made between orthokeratotis, with a granular layer and anucleated squames, and parakeratosis, with nucleated squames. The degree and type of keratinisation show considerable variation (Table 1). In areas not subject to much mechanical stress, such as the cheek, parakeratosis is frequently only demonstrable with special staining techniques, while areas subject to mechanical stress, such as the gums and the hard palate, show orthokeratosis like the epidermis. Particular structural elements in the epithelium are the attachement plaques, or desmosomes. These provide mechanical intercellular cohesion and are responsible for the appearance of 'prickle' cells. The tonofibrils, which act as an 'internal skeleton', and the keratin produced from them afford mechanical protection. Their quantity and density determine the translucency of the epithelial layer and hence the macroscopic coloration

of the mucosa. Increased keratinisation, as in the epithelial hyperplasia known as leukoplakia and in hyperkeratosis, causes the rosy tinge of the mucosa to give way to the naturally whitish colour of the epithelium. Decreased keratinisation e.g.in dysplastic epithelial atrophy, causes dark red discoloration of the mucosa (erythroplakia).

The interdigitation of epithelium (rete pegs) and connective tissue (papillae) also shows considerable variation, being more marked in areas subject to greater wear and tear. The subepithelial connective tissue contains a dense network of capillaries that supply it with blood, and is richly innervated. The muscularis mucosae, which in other mucous membranes separates the lamina propria from the underlying connective tissue is absent.

Both the epithelium and subepithelium tissues contain cells with nonspecific defensive functions(leukocytes) and immunocompetent cells conferring local immunity (lymphocytes and plasma cells in subepithelial tissues; lymphocytes, cerebriform lymphoid cells and Langerhan's cells in the epithelium).The specialised structure of the dorsum of the tongue is well known, and only brief reference need be made to the numerous fine filiform papillae, the scattered funçiform papillae, and the limited number of

vallate papillae arranged at the junction between the anterior two third and the posterior third. In the lateral parts of the posterior third, the presence of lymphoid tissue gives an irregular or nodular appearence.

PRE-MALIGNANT ORAL LESIONS

The term "Precancerous lesion" has been used to signify clinical and/or pathological entities related to cancer development either as a result of prospective study of the progression of such lesions to cancer or a retrospective study of occurrence of such lesions with cancer. In the words of Kramer (1976), "For all practical purposes, a condition may be termed 'precancerous' if it is beleived that the risk of malignent change is sufficiently high to influence the management". In the oral cavity various lesions have been described as precancerous. These are leukoplakia, submucous fibrosis, melanoplakia, erythroplakia, oral lichen planus, papilloma and Plummer vinson's syndrome.

I. LEUKOPLAKIA

Literally the word means a 'white patch'. This is defined as a white patch or plaque on the mucosa that can not be rubbed off and is not ascribable to any other condition (WHO Collaborating Centre for oral pre-cancerous lesions, 1978).

Aetiology and prevalence :

Leukoplakia is said to be associated with various factors such as poor diet, poor oral hygiene, local irritant such as caries, irritation from a badly fitting denture or a broken tooth, oral sepsis, syphilis, tobacco, alcohol, vitamin deficiency, endocrine disturbances, galvanism and actinic radiation in the case of leukoplakia of lips.

Its correlation with the use of tobacco is of particular interest in India where the prevalence and localisation of oral cancer have already been shown to be correlated with the use of tobacco (Orr, 1933; Sanghvi et al, 1955; Wahi et al, 1965; Hirayama, 1966).

The aetiology has got a definite relation with smoking specially in combination with chewing of 'Pan' or 'Betal-nut' (Sugar et al, 1969; Bhonsle et al, 1976; Silverman et al, 1976). Gerry et al (1952) found leukoplakia among 0.2% of 2004 inhabitants, 41% of whom were betal-nut chewers.

Out of 36 cases of leukoplakia studied by Cook (1951), 13 were considered to be related to smoking, 18 were considered to be caused by frictional irritation, and 3 were associated with syphilis.

In India Mehta et al (1961) examined 4734 men of the Bombay police and reported 3.48% oral leukoplakias. 76.5% of the men gave history of Pan chewing, bidi/cigarettes smoking or had both of these habits. The prevalence of leukoplakia was higher among tobacco users (4.5%) than among non-users (0.09%) and higher among those who either chewed Pan (4.2%) or smoked bidi (3.7%). The leukoplakia is common in older age group (Waldron et al, 1975; Silverman et al, 1976). The incidence is slightly more in males than in females. Incidence of malignant changes is more as age advances (Cawson, 1969).

Common sites of occurence of leukoplakia are buccal mucosa (Silverman, 1976). According to Renstrup (1958) buccal mucosa and commisures were most frequently involved, followed in descending order by the alveolar mucosa, tongue, lip, hard and soft palates, floor of the mouth and gingiva. In the study of Shafer and Waldron (1960) the greatest number of cases in both men and women occured on the mandibular alveolar ridge, gingiva or mucobuccal fold. Mehi et al (1961) reported that the buccal mucosa was most frequently involved followed in descending order by along the interocclusal line, angle of mouth, related to last molar tooth, tongue, gums, palate and lips.

Cytological study of leukoplakia :

Exfoliative cytologic studies in oral leukoplakia were first performed by Montgomery and Von Hamm (1951).

Peters and Rijsinghani (1956) studied smears of oral leukoplakia and stated that these smears varied considerably in their cell count, some showing a profuse desquamation of cornified, superficial squamous cells and large number of keratinized cells while other smears showed a varied cell population and considerable cell atypia. Although they were not able to analyze whether the two different smear types could be correlated with the stage of the disease, they were under the impression that early cases of leukoplakia showed the uniform smear type while advanced cases of leukoplakia showed cells of great variation.

According to Sandler and Stahl (1958) oral cytologic technique was well suited for the follow-up of chronic oral lesions such as leukoplakia; yet, in the same year Silverman, et al (1958) stated that "----Unfortunately, suspicious areas of intra-oral leukoplakia prior to fissuring or ulceration, do not lend themselves readily to the cytologic smear method, since only superficial cornified cells are obtainable in smears taken from these white patch areas. They found

no diagnostic change in smears scraped from the nine cases of 'leukoplakia' and stated that leukiplakia remains a diagnostic enigma prior to histopathological examination.

Smears from leukoplakia exhibited acidophilic cytoplasm in approximately 75% of the superficial cells along with cells having pyknotic nuclei and anuclear cells (Wahi and Gupta, 1954).

Umiker et al (1960) reported cytologic atypism in smears from 9 of 45 patient with clinical oral leukoplakia.

Wahi and Luthra (1966) had studied the oral scrape of patients of leukoplakias and reported three types of cellular pattern. First - Simple leukoplakia-associated with ortho-keratotic type of epithelium, smears were showing abundant exfoliation of denucleated superficial cells. The nucleated superficial cells encountered showed minimal amount of nuclear atypism. The second type of smears was accompanied by a parakeratotic type of epithelium, presenting a picture of active leukoplakia with exfoliated cells showing varying degree of nuclear atypism. The third pattern showed a smear with combination of the above two.

Generally, in cases of leukoplakia, the exfoliation of superficial cells pre-dominated the intermediate and basal cells being infrequent. Inflammatory cells are usually abundant and in clumps. Cytoplasmic granulation, vacuolation and peri-nuclear haloes are seen. Macro-nucleoli are observed more frequently. Intermediate and basal cells are seen isolated or in small groups. The cytoplasm is basophilic in most of these cells. An occasional case of leukoplakia with marked cellular atypism shows the exfoliation of one or two benign epithelial pearls.

Shklar et al (1968), Debelsteen et al (1971) do not recommend cytological study from surface smears to determine the pre-malignant nature of leukoplakia. However, Lahiri et al (1974) and Sahiar et al (1975) found the cytological study from surface smears quite an useful method to surveillance for the diagnosis of malignant change in this condition.

Histological study of leukoplakia :

Histologically, leukoplakia, usually show some hyperkeratosis. Commonly there is a epithelial hyperplasia and diffuse chronic inflammatory cells infilteration in the lamina propria. From prognostic point of view special attention is given to epithelial dysplasia. Marked epithelial dysplasia is a fairly reliable indication of impeding serious change.

Lasaks et al (1958) classified leukoplakias into four groups based on histopathological characteristics.

Group I - Epithelial hyperplasia

Group II - Superficial keratosis

Group III - Combination of hyperplasia and keratosis.

Group IV - Epithelial dysplasia

Based on clinical approach Banoczy et al (1972) divided lesions into leukoplakia simplex (having keratinized mucosa), leukoplakia verrucosa (having verrucous proliferation) and leukoplakia erosiva (ulcerated leukoplakia). The type of keratinization varied according to clinical appearance ; hyper ortho-karatosis was found in the majority of leukoplakia simplex and verrucosa and hyperparakeratosis was found in the leukoplakia erosiva cases.They found 12.9% malignant change in erosive, 32% in verrucous leukoplakia and none in simplex leukoplakia group.

II. ORAL SUBMUCOUS FIBROSIS

Submucous fibrosis of the oral cavity, a chronic disease of insiduous onset and unknown aetiology, endemic in India, not seen in the Western countries nor described in literature (The only reports

from other countries are those of Schwartz (1952) who described the entity in Indians settled in East Africa and Su (1954) from Taiwan) is prevalent throughout the Indian subcontinent sparing no caste and creed, affecting the young and the old, the rich and poor alike.The disease is characterised by the presence of palpable fibrous bands in the oral submucosa which may ultimately lead to severe restrictions of the movements of the mouth including that of the tongue.

There was controversy regarding the terminology of the disease. To refer back to our own history of ancient medicine Sushrata in his classification of mouth and throat maladies mentioned a condition 'VIDARI' similar to sub mucous fibrosis (cited by Mukherjee and Biswas 1972). In the modern literature this condition was first reported by Schwartz (1952) in a group of East Indian women residing in Kenya, East Africa as 'atrophica idiopathica (tropica) mucosae'oris'.In India it was first described by Joshi (1953) as submucous fibrosis of the hard and soft palate and pillars. Other names that have been suggested are 'diffuse oral submucous fibrosis' by Lal (1953), 'idiopathic scleroderma of the mouth' by Su (1954), 'idiopathic palatal fibrosis' by Rao (1962) and 'sclerosing stomatitis' by Behl (1962) and 'Juxta epithelial fibrosis' by Pindborg (1966). The term 'oral sub mucous fibrosis' is now widely accepted.

Geographical Distribution

Oral sub mucous fibrosis has been recorded mainly amongst Indians but occasional cases have been reported from Taiwan (Su, I.P., 1954), Nepal, Thailand, South Vietnam and Ceylon (Pindborg and Sirsat, 1966). Among Indians living outside India submucous fibrosis has been found in Malaysia (Pindborg and Sirsat,1966), Uganda (Millard, 1966) and South Africa (Dockrat and Shear, 1964). Isolated cases among Pakistanis and Indians living in the United Kingdom have also been reported (Rees and Madan, 1968). Furthermore, oral submucous fibrosis (OSMF) has been diagnosed among domiciled European living in Hyderabad and a British female living in England and married to a Pakistani (Simpson, 1969) Dockrat and Shear (1969) examined 1000 Indians in South Africa and found a prevalence of 0.5%.

In India oral sub mucous fibrosis cases have been reported from different regions - From Madhya Pradesh (Lal, 1952); Bombay (Joshi, 1953; DeSa, 1957) ; Bihar (Sharan, 1959); Hyderabad (Rao, 1962) and from Gorakhpur, Utter Pradesh (Gupta et al, 1978).

Prevelence and Incidence

Epidemiological studies on prevalence of oral sub mucous fibrosis have been carried out by various

investigators. Pindborg et al (1964) examined 35,000 urban Indians seeking admission in clinics at Dental Colleges in Lucknow, Bombay, Banglore and Trivandrum and found the prevalence of 0.5%, 0.5%, 0.2% and 1.2% respectively. The prevalence rate in Southern India is found to be more (Pindborg et al, 1964; Wahi et al, 1966; Mehta et al, 1971). Pondborg (1980) has estimated that not less than 2,50,000 cases of oral sub mucous fibrosis (OSMF) are present in India. Varghese et al (1986) have found an increased prevalence of OSMF in the cashew workers of Kerla (7.85%).

Reports of sex ratio vary, however the majority demonstrate a female predominance (Schwartz, 1952; Rao and Raju, 1954; Rao, 1962; Wahi et al, 1966; Pindborg et al, 1968). The largest number of cases occur between the ages of 20 and 40 years (Wahi et al 1966, Zachariah et al 1966 and Mehta et al, 1971). DeSa (1957) and Sirsat and Khanolkar (1962) found a nearly equal distribution of cases among males and females. Sharan (1959), Su (1954) found a predominance of males in their study.

Aetiological Factor

The exact aetiology is not yet established. Phatak (1978) found significantly elevated levels of globulins and immunoglobulins and suggested it to be

an autoimmune disease. Various causative factors have been mentioned such as chewing of tobacco, betal nut, pan and pan masalas, eating of spices and chilis, hereditory predisposition, Vitamin A and B complex deficiency, localised collagen disease and reaction to bacterial infection etc.

Tobacco, Betel nut, Pan and Pan Masalas chewing :

The chewing of betal quid is mentioned in the Sanskrit 'Sushrata Samhita' believed to have been written about 600 A.D. near Banaras (Varanasi). The Sanskrit for the leaf of betel vine 'Tambula' persists in the modern Hindi 'Tambuli' and in the Arabic and Persian 'Tambula'.

The role played by tobacco is debatable as submucous fibrosis had occured in patient who had never indulged in tobacco habits (Paymaster 1956). Betel nut chewing causes degenerative changes in connective tissue of oral mucosa followed by fibrosis (Sharan, 1959; Lal, 1953 and Lanner and Shear, 1969). The disease according to Su (1954) in the betel nut chewers may be caused by-

(i) Amount of tannic acid (14 to 18%)

(ii) Influence of slaked lime

(iii) Continued and prolonged action of alkaloid arecoline on nerve ending and consequent neurostrophic disorders.

According to Pindborg (1965) the most important aetiological factor for producing oral submucous fibrosis and oral cancers are tobacco and betel nut. Caniff and Harvey (1981) proved that areca nut extract can act as a potent stimulator of collagen synthesis in human fibroblast culture. Mejhji et al (1982) showed that the tannins present in areca nut reduced the degradation of collagen by collagenase. Chewing of pan which consists of the ripe betel leaf coated with crude lime, sprinkled with powdered acacia catechu, containing small pieces of areca catechu nut, few dried leaves of anethum graveolens with or without tobacco. Some people chew pieces of areca catechu alone or scented supari, others chew crude tobacco or tobacco mixed with lime which is usually placed in the vestibule of the mouth for slow absorption (DeSa 1957). The habit of chewing of Pan Masalas are of recent origin. All varieties of Pan Masalas contain nearly all the constituent of betel quid except betel leaves. However, Varghese et al (1986) reported that arecoline plays no significant role in the causation of human submucous fibrosis as there were six patient studied by them who had never taken Pan, betel or tobacco any time in their life.

Dietatic Habit

Since the disease occurs predominantly among Indians and peoples of Indian origin, a possible cause has been suspected in their common diet. Spices like pepper and chillies (capsicum annum and capsicum frutescens) being an essential ingredient in Indian diet, are universally used in all parts of India to season food. Support for this theory, that chillies also may be a causive factor, is found in the occurrence of submucous fibrosis among Indians living outside India but maintaining Indian dietary habits. An allergic reaction has been suggested as the possible cause of oral submucous fibrosis (OSMF) (Sirsat and Khanolkar, 1962; Pindborg et al, 1968), the possible allergen which has been suspected, common in the Indian diet, is chillies (Hammer et al, 1974).

Collagen Disease

Clinical picture of stiffness and immobility of oral mucosa and histological changes in connective tissue suggested a collagen disease (DeSa, 1957; Moss and Madan, 1968).

According to Rao, A.B.N. (1961) it will be more logical to group this condition with localised forms of collagen diseases such as Peyronie's disease.

Dupuytren's contracture, Keloids, idiopathic retroperitoneal fibrosis (Raper, 1960) and idiopathic mediastinal fibrosis (Barrett, 1958).

Genetics :

Genetic factors might play some role in genesis of oral submucous fibrosis (Hammer et al, 1971).

Oral Infections :

The role of oral infections as a factor for causing oral submucous fibrosis has been emphasised (DeSa, 1957).

Vitamin Deficiency :

Wahi et al (1960) had suggested the Vitamin A and B deficiency associated with tobacco chewing may be the possible cause.

Symptomatology :

Earliest symptoms are burning sensation of the oral mucosa, inability to eat spicy food, stomatitis, dryness of the mouth or excessive salivation, vesicle formation and ulcerations. Later stiffness of certain areas of the oral mucosa result in inability to open the mouth completely, to protrude the tongue, to whistle

or to blow out a candle, trismus, refered pain in the ears, deafness or nasal voice may be observed in some cases.

Clinically the disease may be divided into three stages, in which the patient present themselves for treatment; Stage I - Stomatitis or Vesiculation; Stage II - Fibrosis; Stage III - Sequlae (DeSa 1957).

Oral submucous fibrosis as a precancerous condition

The possible precancerous nature of submucous fibrosis was first mentioned by Paymaster (1956) who described the development of slow growing squamous cell carcinoma in a third decade of the one third patients with submucous fibrosis. Sirsat and Khanolkar (1962) has reported malignancy in four out of 85 cases (4.7%). Pindborg (1965) demonstrated that Indian patients with submucous fibrosis have a Higher incidence of leukoplakia and of carcinoma than those without the disease. In subsequent study Pindborg (1965) has himself reported 40 cases of submucous fibrosis among 100 Indians with oral cancer. Wahi et al (1966) had found cancerous change in 3% of cases.

Histopathological changes

Sharan (1959), Rao (1962), Sirsat and Khanolkar (1957 and 1962), Wahi (1965) have described the histological

changes found in submucous fibrosis. Histologically most of the cases are characterised by atrophy of the epithelial layer with loss of rete pegs. Epithelial atypia is also present in a few of the cases. The underlying connective tissue shows severe hyalinization with homogenization of collagen bundles. Fibroblasts are markedly diminished and blood vessels are completely obliterated or narrowed. Some chronic inflammatory cells infiltration is also present.

Cytological study of submucous fibrosis

Wahi and Luthra (1966) had studied the oral scrapes of patients of oral submucous fibrosis and reported that smears from these cases showed a preponderence of superficial cells while intermediate and parabasal cells were less frequent.

In these smears they found that the anucleated squamous cell were isolated or in clusters. Frequently the cytoplasm was eosinophilic and occasionally it was intensely orangophilic. Sometimes these cells showed the presence of 4 to 10 brown to black cytoplasmic granules. A majority of the cells showed a cyanophillic cytoplasm with vacuolation and perinuclear haloes. The nuclei were round to oval with distinct nuclear membrane, prominent nucleoli and the chromatin presented a peculiar 'rarified' pattern.

III. LICHEN PLANUS :

It literally means a cryptogenic mass like plant (algae and fungi mixed) forming patches on rocks or tree trunk.

It is generally discovered only by accident, but often the patient will complain of discomfort or soreness of oral mucosa (Shklar and McCarthy, 1961 ; Kramer et al, 1970). Mucosal lesions are usually multiple and often have a symmetrical distribution. They commonly take the form of minute white papules which gradually enlarge and coalesce to form either a reticular, annular or plaque pattern. A characteristic feature is the presence of Slender white lines (Wickham's Striae) radiating from the papules. The plaque form may be difficult to distinguish from leukoplakia, but in lichen planus there is usually no change in the flexibility of the affected mucosa. In some patient the lesions are atrophic, with or without erosions. Oral lesions of lichen planus may also include bullae, but these are rare (Kramer, 1978). Histologically there is hyperkeratosis or parakeratosis and thickening of the granular layer, acanthosis with intracellular oedema of the spinous cells, 'saw-tooth' appearence of the rete pegs is less frequent, necrosis or liquefaction degeneration of the basal layers of cells with the appearence of a thin band of eosinophilic

co-agulum in the place of this basal layer and finally, infilteration of lymphocytes and only occasional plasma cells into the sub-epithelial layer of connective tissue (Shafer - A text book of oral Pathology, 1967).

Many earlier reports suggest the premalignant nature of oral lichen planus (Cawson, 1968). Carcinoma may arise in oral lichen planus but does so only rarely (Shklar and McCarthy, 1961; Kovesti and Banoczy, 1973), and there is greater risk when lichen planus is in the atrophic or erosive form (Kramer, 1976).

Silverman (1974) found average age of onset to be slightly over 50 years. In his study, 65% cases were women and majority of them had egosive type of lesions. Skin involvement was only in 28%. There was no apparent causative factor. In a study of Bhonsle (1976) amongst reverse 'dhumti' smokers of Goa, India, the incidence of Lichen Planus of the mouth was 0.2%.

According to Wahi et al (1966) the smears of the patients of Lichen Planus are characterized by abundant exfoliation of epithelial and inflammatory cells. Nucleated superficial cells predominate. Cytoplasmic vacuolation and nuclear fragmentation are prominent features. Leukocytic inclusions are seen in

some pre-cornified and cornified cells. The chromatin pattern is a predominently finely granular. Intermediate and basal cells occur in small groups or isolated. These are markedly enlarged and show large cytoplasmic vacuoles. The nuclei are round to oval and show distinct nuclear membrane. Few basal cells show multi-nucleation (3-5 nuclei).

IV. ERYTHROPLAKIA :

Erythroplakia is defined as a brilliant, dark red circumscribed lesions that can not be rubbed off and is not ascribable to any other definitive condition. It presents as velvety red patches that slowly spreads. The margin of such a lesion may also show whitish (leukoplakic) changes. Usually the epithelium is thinner than normal and may show typical change of carcinoma-in-situ (Shafer, 1975). Erythroplakia is less common than leukoplakia and has, on the whole, greater malignant potential. Kramer (1973) has said that erythroplakia should be regarded as carcinoma until proved otherwise.

Cytological findings in cases of melanoplakia and erythroplakia are similar to leukoplakia in cell type and chromatin pattern. No melanin pigment containing cells are seen in cases of melanoplakia (Wahi et al, 1966).

VI. PLUMMER VINSON SYNDROME :

The plummer-vinson syndrome (also known as Paterson-Brown-Kelly syndrome), a form of iron deficiency anaemia was first described by plummer in 1914 and by Vinson in 1922 under the term "hysterical dysphagia". Ahlbom (1936) defined it as a pre-disposition for the development of carcinoma in the upper alimentary tract. It is an established precancerous condition for causation of post cricoid carcinoma in females, specially in Western countreis. However, its association with oral cancer also was shown by Wynder et al (1957) in Sweden.

It occurs chiefly in women in the fourth and fifth decades of life. Presenting symptoms are cracks of fisures at the corners of mouth, a lemon tinted pallor of skin, a smooth, red, painful tongue with atrophy of the filiform and later the fungiform papillae, and dysphagia. The mucous membrane of the oral cavity and oesophegus are atrophic and show loss of normal keratinization. Monto and his associates (1961) reported unusual alterations in exfoliated squamous epithelial cells of the tongue in cases of severe iron deficiency anaemia. These changes consisted of a deficiency of keratinized cells, a reduced cytoplasmic diameter of cells with a paradoxical enlargement of the nucleus, and abnormal cellular maturation characterized by a disturbed nuclear pattern, an increase in nucleoli, presence of double nuclei and karyorrhexis.

VII. STOMATITIS NICOTINA (LEUCOKERATOSIS NICOTINA PALATI, NICOTINIC STOMATITIS) :

It is already established that many habits produce epithelial changes in the buccal mucosa. Sirsat et al (1974) found that tobacco taken in any form, produces a more profound degree of keratinization. The habit of reverse smoking, prevalent in certain parts of India, produces an extensive hyperorthokeratosis often associated with epithelial atypia of the palatal mucosa (Mehta et al, 1969; Pindborg et al, 1971). Reddy (1974) in his studies of cancer of the palate among reverse smokers, found the association of stomatitis nicotina, a papular umblicated lesion in the glandular zone of the hard palate to be premalignant.

In the early stages of stomatitis nicotina, the mucosa is reddened, but it soon becomes greyish white and may present a wrinkled appearence. Later it becomes thickened, and white umblicated nodules with red centres appear. Histopathologically the epithelium shows acanthosis and hyperorthokeratosis and hyperparakeratosis is seen around the orifices of the ducts of the palatal mucus glands. The epithelium linining often shows squamous metaplasia. There is usually a moderate degree of chronic inflammatory infilteration in the subepithelium connective tissue and around the gland acini.

Stomatitis nicotina is often a reversible condition that is resolved when smoking is discontinued (WHO collaborating centre for oral pre-cancerous lesions, 1978).

VIII. ORAL CANDIDOSIS :

Eyer and Nally (1971), while presenting three cases of chronic oral candidosis observed eventual malignant change in two, and opined that there is a definite propensity for malignant change in these lesion.

In the chronic infection there may be gross epithelial hyperplasia. A moderate degree of epithelial dysplasia is often seen, but there is evidence that this may regress if the candidal infection is eliminated. However, there is also some evidence to suggest that malignant changes are more likely to occur in chronic candidal leukoplakia than in non-candidal leukoplakia, the inter-relationship between candidal infection, the epithelial dysplasia and the risk of future malignancy remain uncertain. (WHO Collaborating Centre for Oral precancerous lesions, 1978).

IX. DENTAL AND ORAL INFECTION :

Lash et al (1961) among many others, found a common association of oral sepsis with carcinoma of the tongue. However, Cade and Lee (1957) observed healthy oral mucosa associated with the same disease. Though none yet has been able to show definitely an association of dental irritation and trauma as significant factors in oral cancer, Wood (1961) considers the rising standard of oral hygiene to be a possible factor in decreasing the death rate from oral cancers in Western countries.

X. SYPHILIS :

Wynder et al (1957) found that syphilis was shown to be of some importance in the development of cancer of the lip and of the anterior two third of the tongue. It can not be established whether this relationship is due to syphilitic glossitis or to arsenical therapy, which most of these patients have received. At any rate, with the modern methods for control of syphilis through its early treatment with antibiotics, this factor will be less important.

MALIGNANT ORAL LESIONS :

Malignant lesions of the oral cavity originate from epithelial tissues or the mesodermal elements. Carcinomas, i.e. malignant lesions of epithelial origin is the commonest variety encountered and of these, squamous cell carcinomas are the commonest. Pathological aspects of the lesions in various sites are described below :-

Carcinoma of the lip :

The incidence varies in different parts of the globe. It is more in males. Spitzer et al (1975) in a study found that, despite the effect of pipe smoking, outdoorness and age on the lip cancer in general, the occupation of fishing is an additional independant risk. Khanolkar (1959) observed a high frequency of carcinoma of the lower lip, specially in males in Bihar and adjacent areas of Uttar Pradesh, India, where the habit of keeping 'Khaini', quid of powdered tobacco and slaked lime in lower gingivolabial sulcus for many hours is common.

The lesion starts as a small warty growth, ulcer or a fissure on the mucosal surface of the lip. Ulcerative forms progress relatively rapidly and invade deeper tissues and adjacent structures early.

Lymphnode metastasis occurs relatively late and when occurs, submental and submandibular nodes are the first to be involved with subsequent extension to the upper deep cervical nodes. Histologically majority of the tumours are well differentiated.

Carcinoma of the buccal mucosa :

"Carcinoma of the buccal mucosa overshadows all other type of oral cancers in the South-Western Coastal regions of India" - Baruah (1964). The term buccal mucosa here indicates the mucosa of the buccal aspect of the cheek. Carcinoma usually starts in the region opposite the lower third molar tooth. It may also start as a malignant transformation of the pre - existing leukoplakic patch. The tumour starts as a small nodule, enlarges to form a wartlike growth and then ulcerates. The lesions may, however, start as an ulceration. Extension to surrounding areas takes place leading to trismus, dysphagia and various other manifestations. Metastasis reach to submandibular and upper deep cervical nodes. Histological picture in majority of cases shows a well differentiated squamous cell carcinoma.

Carcinoma of the Gum :

Carcinoma of the gum may arise from two sources, the common epidermoid carcinoma from the mucosa and carcinoma from minor salivary glands present in the alveolus.

The carcinoma occurs generally in the pre-molar and molar regions and common site of occurence is the lower alveolus. Cooke (1976) attributed the occurence to chewing of betel nut with tobacco and slaked lime and the anatomical flow of saliva. Smoking is another important aetiological factor (Cady and Catlin, 1969). Males are more affected than females.

The growth may be ulcerative or papillary, with the former having more tendency to invade the underlying tissues and bone at an early stage. However, cancers from minor salivary glands, though commonly present as non-ulcerated masses frequently invade the underlying bone (Cady and Hutter, 1969). Microscopically carcinoma gingivae is practically always a well differentiated variety. Metastasis takes place early, more in cases of lower gum to submandibular nodes and then to the cervical nodes.

Carcinoma of the floor of the mouth :

The relatively slowing growing carcinoma generally occurs in the anterior portion of the floor of the mouth away from the midline, in the region of the junction with the tongue. The tumour may present as a wartlike growth and remain superficially, may be ulcerative or even may remain in the submucosa presenting

as a fissure in the oral cavity. Lymph node metastasis occurs in most cases to submandibular and submental nodes. Histologically most of them are well differentiated.

Carcinoma of the hard palate :

Carcinoma of the hard palate is prevalent in certain districts of Andhra Pradesh, India, where the habit of reverse smoking of local cigars 'Chutta' is common. The tumour may be of a papillary variety or an ulcerative growth invading the under-lying bone and thus, may cause perforation of the palate. Eneroth et al (1970) showed a high incidence of mucoepidermoid carcinoma occuring from the covering epithelium of the terminal portion of the ducts of the minor salivary glands. There is both mucous secreting cell proliferation and epidermoid differentiation. The poorly differentiated variety is highly malignant. Reddy et al(1974) observed a high incidence of carcinoma palate in females reverse smokers in the posterior half of the hard palates away from the midline where there is the highest concentration of glands. Lymph node metastasis occurs in 30% cases (Lucas, 1964).

Carcinoma of the anterior two-thirds of the tongue :

The lesion shows a higher sex incidence in male and occurs mainly in middle and old age. Chronic

irritation and syphilis, if present may play a part in its occurence. Hyperkeratosis or leukoplakia is a precursor. The lesion may present initially as a small papilloma or a warty growth or as an ulcer with everted edges. The usual site of occurence is lateral border of the middle third of tongue, microscopically, usually an epidermoid carcinoma showing higher grades of differentiation. Infilteration to submandibular and submental nodes is rapid, leading to fixation of the tongue. Prognosis is comparitively poor.

Some uncommon malignant tumours of oral cavity :

Verrucous carcinoma :

A slow growing carcinoma, occurs chiefly in older age group above the age of 60, and affects the buccal mucosa, gingiva, palate, tongue and tonsils. The lesion is a papillary mass composed of heaped up folds of tissue. Though characteristically indolent, local infiltration may occur upto bones. Lymph node metastasis never occurs, though there may be inflammatory enlargement of regional lymph nodes. Microscopically, it is always well differentiated with intact basement membrane making the diagnosis of carcinoma difficult. Prognosis with proper treatment is excellent.

Malignant melanoma :

Also known as melanocarcinoma, it is a rare tumour in this region. The tumour arises from melanoblasts and oral melanosis may be a precursor. The biological characteristics of the tumour, in this region, have a great tendency to infiltrate the adjacent tissue structures and a greater disposition to metastasis (Raicev and Buryak, 1970). It may progress, unnoticed, in the oral area until it reaches a considerable size (Shimada, 1976). Soman and Sirsat (1974) presenting a series of 24 cases of malignant melanoma in Indians, found a high incidence in males. The common site of affection was the alveolus and palate.

Malignant connective tissue tumours :

Malignant connective tissue tumours in the oral cavity are very rare. Various types such as fibrosarcoma, lymphsarcoma, oesteogenic sarcoma, reticulum cell sarcoma, liposarcoma have all been reported.

Multicentric oral carcinoma :

Oral carcinoma may be multicentric in origin particularly in heavy smokers. This may be due to the abnormal state of the oral mucosa for a longer period of time under the influence of chronic irritation prior to the development of overt carcinoma (Slaughter

and colleagues, 1946, 1953). Oral carcinoma may also be associated with primary carcinoma elsewhere in the body. Two or more oral carcinomas or additional malig- lesions in the pharynx, oesophagus and other structures have been reported (Moertal et al, 1958; Sharp et al, 1961; Meyer and Shklar, 1960).

Malignant tumours of the Oropharynx :

Malignant lesions may occur in any parts of the oropharynx and like the oral cavity, in this region also, squamous cell carcinoma is by far the commonest variety of malignancy encountered.

Carcinoma of the soft plate and fauces :

Carcinoma of the soft plate and uvula is commoner than that of the hard palate. Posterior border is the usual site to start with. Seydel and Scholl (1974) in their study, found that the lesion occurs in male after fifth decades of life. Carcinoma highly differentiated is usually of epidermoid variety (Hjertman and Emeroth, 1970). Local spread takes place to the pterygoid fossa, hard palate etc. leading to trismus, dysphagia. Metastasis takes place to upper deep cervical nodes. There is often bilateral involvement of the nodes in central soft palate lesions.

Malignancy of the tonsils & malignancy of the base of tongue :

They are carcinoma, lymphoepithelioma and lymphosarcoma of which carcinomas are the commonest. Carcinoma of tonsil is one of the common variety of oropharyngeal malignancies. The lesion is uncommon before the age of 50 years and the incidence is higher in males. It is more prevalent in heavy smokers, heavy drinkers and those who have poor oral hygiene (Fleming et al, 1976). The lesion may be proliferative or ulcerative.

Malignancy of the base of the tongue :

Malignancies of the base of the tongue are also carcinomas, lymphoepitheliomas and lymphosarcomas, of which carcinomas are the commonest. Pathologically it is similar to malignancy in the tonsil. But, because of involvement of the tongue musculature, there is alteration of speech, the so-called 'hot potato voice' (DeWeese and Saunders, 1973). There is often bilateral involvement of upper deep cervical nodes.

Malignancy of the oropharyngeal wall :

Most of the tumours are carcinomas showing a high degree of malignancy. The growth may be proliferative or ulcerative showing low grades of differentiation. Regional lymph node metastasis is early and bilateral involvement takes place in the posterior pharyngeal wall lesions. Overall prognosis is poor.

AETIOLOGICAL FACTORS IN THE GENESIS OF CARCINOMA OF THE ORAL CAVITY :

The aetiology of oral carcinoma is as debatable and diverse as any other aspect of the condition. Various aetiological factors suspected to be related to cancer of oral cavity are as follows :-

Tobacco :

The high incidence of oral cancer in India has been associated with tobacco chewing and/or tobacco smoking habits (Khanolkar, 1944, 1959; Sanghvi et al, 1955; Shanta and Krishnamurti, 1963; Paymaster, 1971; Wahi, 1968; Reddy et al, 1975; Khanna et al, 1975).

The effect of smoking are partly the result of the heat generated and partly due to the chemical composition of fumes. It is fully established that tobacco products are carcinogenic. Kerr (1948) states that nicotine along does not change or cause the condition. Tars contain Benzpyrene which produces carcinoma in experimental animals. The effect on the mucosa usually occurs in a heavy smokers. If the pipe or cigarette is habitually held in one position, only the area on which the smoke cheifly impinge, may be involved (McCarthy, 1936).

Wynder and Bross (1957) have reported that the smoking chiefly of cigarette and pipes is responsible for oral cancer while cigrette smoking causes mainly lung cancer.

In the series of Wahi et al (1958), 88 cases in a total of 750 were smokers, out of which 64 cases were associated with tobacco chewing. They are of the view that in our country smoking of cigarette and bidis is less responsible for oral cancers than the tobacco chewing. It is possible that smoking continued over a long periods may contribute to the development of cancer of the lip and tongue due to combined action of heat and tobacco carcinogens. 74% of patient who chewed tobacco had cancer on the side on which they kept the quid (Chawla et al, 1969). Jussawala and Desh Pande (1971) made a retrospective study of cancer at high risk sites at Bombay Registry and found chewing and smoking of tobacco as aetiological factor of oral, pharyngeal, laryngeal and oesophageal cancers. The risk of developing cancer in buccal mucosa was found to be 7.7 times higher in chewers than in non-chewers.

Alcohol :

Wynder and Bross (1957) reported that alcohol has a marked influence on the development of cancer of the mouth. This could be due to a direct effect of the

alcohol on the tissues or may be due to a decrease of protective saliva from the mucosa, which would tend to make it more susceptible to the effect of tobacco. A third possibility is an indirect action due to nutritional deficiencies. Alcoholics develop deficiencies of ascorbid acid, thiamine, riboflavine, and upon liver involvement, also of vitamin A (Jellinck and Jolliffe, 1940).

Cade and Lee (1957) found that heavy alcohol consumption particularly whisky to be of a significant factor among patients of carcinoma of oral cavity.

Dental conditions and poor oral hygiene :

Trauma and irritation due to ill fitting dentures or a sharp jagged tooth also predisposes to the development of oral cancer. Khanolkar (1944) notes that chronic irritation from a jagged tooth has no part in the causation of cancer in Bombay but Balendra (1949) considers that it is an important factor.

A poor oral hygiene is commonly met in all the cancer patients in India. There is tartar deposition over the teeth and advanced pyorrhoea usually in all cases of oral cancer (Wahi et al, 1958).

Nutritional Deficiency :

The frequency of oral carcinoma shows a close relation to the economic status which in turn indirectly related to quality of the food of the individuals. Most of the patients suffered from mild to severe degree of avitaminosis (Orr, 1933; Wahi, 1958).

Martin and Koop (1942) have stressed that malnutrition and vitamin B-complex deficiency with vitamin A deficiency are probably associated with oral cancer.

Social Status :

Carcinoma of the oral cavity is found to be more in individuals of the labour and lower middle class group (Wahi et al, 1965). This may be explained as a result of poor oral hygiene and gross oral sepsis in this class of people.

Syphilis :

Syphilis was shown to be some importance in the development of cancer of the lip and of the anterior two thirds of the tongue (Wynder et al, 1975).

GEOGRAPHICAL DISTRIBUTION AND INCIDENCE :

International Union against cancer (U.I.C.C., 1970) in its publication reported higher incidence of cancer of mouth, tongue and pharynx in India in relation to other countries (cited from Agarwal et al, 1985).

Cancer of the oral cavity is common in India, where it accounts far 40% of all male cancer admissions to the Tata Memorial Hospital, (Khanolkar, 1950).

Dr. David Branes (1980), Chief of the Oral Health Programme of W.H.O., has also indicated in a report that the incidence of mouth cancer is highest in India, being 35 to 40% as compared to only 3 to 5% in North American and European countries (cited from Agarwal et al, 1985).

A statistical survey was undertaken by Kini and Subba Rao (1937) as regards the cancer palate, which was prevalent in the women of Vishakhapatnam District of Andhra Pradesh. It is due to peculiar habit of reverse smoking of cigar (Chutta).

SITES OF INVOLVEMENT IN ORAL CAVITY :

The cheek and other unspecified part of mouth were found to be more commonly involved (Wahi et al, 1965 from Agra, U.P. and Gangadharan, 1979 from Kanpur), followed by tongue. However, Jussawalla (1980) has reported the reverse i.e., hypopharynx to be commoner than cheek, in the Bombay Cancer Registry.

TABLE NO. II

Site wise frequency of oral cancer as reported by different observers.

Name of Authors	Total cases	Lips %	Buccal mucosa %	Tongue %	Gingivae %	Palate %
Wahi et al, 1965 Agra (India)	1916	2.6	52.3	26.9	10.2	5.9
Khanolkar, 1946 Bombay (India)	1000	1.7	16.5	52.2	6.0	6.2
Khanolkar and Suryabai, 1945 Vishakhapatnam (India)	285	7.0	15.4	27.7	4.9	36.8
Somervell, 1944 Travencore(India)	3397	6.0	45.5	13.0	35.0	-
Haldar, P.K. 1949 - 1952 Agra (India)	600	3.4	54.1	26.0	8.3	8.2

AGE :

Incidence of oral cancer was more common in persons above 50 years age (Orr, 1933; Paymaster, 1957).

SEX :

The oral cancer is more prevalent in males as compared to females, the ratio being 2:1 (Wahi et al, 1958).

In Sweden, cancer of the tongue, gum and buccal mucosa is about as frequent in women as in men, due to prevalence of Plummer-Vinson disease among Swedish women (Wynder et al, 1957).

RELIGION :

Wahi et al (1952) reported that in their series Hindus were affected 1.8 times more than Muslims.

ORAL AND OROPHARYNEAL TUMOURS :

(International Histological Classification of Tumours, W.H.O.).

I. TUMOURS OF SQUAMOUS EPITHELIUM

(A) BENIGN

1. Squamous cell papilloma

(B) MALIGNANT

1. Intraepithelial carcinoma (Carcinoma in situ)
2. Squamous cell carcinoma
3. Variants of squamous cell carcinoma
   (a) Verrucous carcinoma
   (b) Spindle-cell carcinoma
   (c) Lymphoepithelioma

II. TUMOURS OF GLANDULAR EPITHELIUM

III. TUMOURS OF SOFT TISSUES

(A) BENIGN

1. Fibroma
2. Lipoma
3. Leiomyoma
4. Rhabdomyoma

5. Chondroma

6. Osteochondroma

7. Haemangioma

   (a) Capillary

   (b) Cavernous

8. Benign haemangioendothelioma

9. Benign haemangiopericytoma

10. Lymphangioma

    (a) Capillary

    (b) Cavernous

    (c) Cystic

11. Neurofibroma

12. Neurilemoma (Schwannoma)

(B) MALIGNANT

1. Fibrosarcoma

2. Liposarcoma

3. Rhabdomyosarcoma

4. Leiomyosarcoma

5. Chondrosarcoma

6. Malignant haemangioendothelioma (angiosarcoma)

7. Malignant haemangiopericytoma

8. Malignant lymphangioendothelioma (lymphangiosarcoma).

9. Malignant schwannoma

IV. TUMOURS OF THE MELANOGENIC SYSTEM

(A) BENIGN

Pigmented naevus

Non-pigmented naevus

(B) MALIGNANT

Malignant melanoma

V. TUMOURS OF DISPUTED OR UNCERTAIN HISTOGENESIS

(A) BENIGN

1. Myxoma

2. Granular cell tumour (Granular cell "Myoblastoma").

3. Congenital "Myoblastoma"

(B) MALIGNANT

1. Malignant granular cell tumour (Malignant (nonorganoid) granular cell "Myoblastoma").

2. Alveolar soft-part sarcoma (Malignant organoid granular cell "Myoblastoma").

3. Kaposi's sarcoma

VI. UNCLASSIFIED TUMOURS

VII. TUMOUR LIKE CONDITIONS

1. Verruca vulgaris

2. Papilliferous hyperplasia

3. Benign lymphoepithelial lesion

4. Mucocele

5. Fibrous overgrowth

6. Congenital fibromatosis
7. Xanthogranuloma
8. Pyogenic granuloma
9. Peripheral giant cell granuloma (giant cell epulis)
10. Traumatic neuroma
11. Neurofibromatosis

CARCINOMA-IN-SITU :

Carcinoma-in-situ (Intra-epithelial carcinoma) is characterised by an epithelium that manifests morphologic malignancy but does not demonstrate invasion of the underlying connective tissue.

It is characterized by marked cellular pleomorphism and by loss of polarity and surface stratification, with the whole thickness of the epithelium showing malignant cellular features. The basement membrane is intact. Nuclei are hyperchromatic and show wide variation in size and shape. The nucleocytoplasmic ratio is altered. Chromatin is either finely granular, or coarsely clumped and irregularly distributed. Nucleoli are enlarged and often multiple. Mitoses, often abnormal, occur in all parts of the epithelium. Subepithelial tissues commonly show chronic inflammation and increased vascularity.

CYTOPATHOLOGICAL ASPECTS OF MALIGNANCY :

A malignant cell is a modified normal cell. It varies from the normal cell in many aspects. The criteria for malignancy was studied by many authors. These criteria are present in individual cells, the cells in clusters and also there are certain indirect ways of diagnosing malignancy by cytological study.

THE CRITERIA OF MALIGNANCY IN A SINGLE CELL : They are as follows :-

1. Enlargement of nucleus

It is agreed that the nuclear enlargment is because of an increase in the D.N.A. content in the malignant nucleus.

2. Alteration of nuclear - Cytoplasmic ratio (N/C ratio)

In normal cells the ratio between the volumes of the nucleus and cytoplasm remains within a normal limit. However, there may be an overall increase of nucleus and cytoplasm volumes following irradiation, inflammation etc. keeping the N/C ratio within the normal limit. In malignant cells, the nuclear volume is more with proportional decrease of the volume of cytoplasm. In poorly differentiated cells the nucleus may cover almost the whole of the cell keeping only a rim of cytoplasm around it.

3. Hyperchromatism of the nucleus -

The D.N.A. content in malignant cells is both increased and widely distributed. This is responsible for hyperchromatism of the nucleus with basic dyes.

4. Coarsely granular clumping of chromatin -

Unlike evenly distributed chromatins in the nucleus of normal cells, in cancer cells, chromatins reveal a coarsely granular or thick strandlike distribution. The space in between the chromatin clumps seen to be free of chromatin particles.

5. Thickness and irregularity of nuclear membrane -

This is produced by chromatin condensation at the nuclear membrane. The infoldings and irregular indentations are better visualised under electron microscope.

6. Prominence and multiplicity of nucleoli -

Prominence of nucleoli is because of relative increase of chromatins in the nucleoli. Also there is increase in size and number.

7. Abnormal mitosis -

Abnormal and frequent mitosis are indicative of malignancy although it is not usual to see it in normal cells.

8. Multinucleation and multilobulation -

Because of abnormal mitosis, there is marked indentation and wrinkling of nuclei. However, it may be seen in benign cells like mesothelial, transitional variety etc. In such cases other characteristics of malignancy helps in contributing to the diagnosis.

9. Variation in nuclear and cytoplasmic shape and size -

There is remarkable variation in size and shape of nuclei and cytoplasm when compared amongst one another in individual malignant cells.

CRITERIA FOR MALIGNANCY IN A CLUSTER OF CELLS : They are described below :-

1. Anisokaryosis and cell clumping with pleomorphism -

Malignant cells tend to exfoliate in clumps and in these clumps, marked variation in size and shape of nuclei can be noticed as an important feature of malignancy.

2. Irregular arrangement of cells -

There is irregular piling up of a clump of cells which is comparable to loss of polarity in the histological picture.

3. Pair cells and inclusion cells -

Because of incomplete division, abnormal mitosis, two malignant cells may be connected together at a portion of cytoplasm forming 'pair cells'. Because of the same phenomenon, one malignant cell may be found completely inside another malignant cell as an 'inclusion cell'.

'DYSKARYOSIS' IN CYTOLOGICAL STUDY :

The term 'dyskaryosis' means abnormal hypertrophy of the nucleus, while the cytoplasm is well differentiated and is meant to denote abnormality in the cells in smears within benign limits. There is nuclear enlargement, hyperchromatism, irregularity of nuclear rim and multinucleation. The grades of dyskaryosis is from 'mild' to 'severe' according to the degree of nuclear abnormality.

'Mild' dyskaryosis shows slight nuclear hypertrophy, mild hyperchromatism and somewhat coarse distribution of chromatin. The cytoplasm is fairly well differentiated showing the maturity of the cells.

'Severe' dyskaryosis shows the nuclear changes closely mimicking those in the malignant cells. There are prominent nucleoli, clumping of chromatins, irregular infoldings of nuclear rim etc. Cytoplasm is not always well differentiated having an indistinct cellular border.

CELLULAR CHANGES MIMICKING MALIGNANCY :

1. Cellular changes during inflammation -

Active proliferation of cells may take place as a direct response to an injurious agent. There may be hyperchromatism, multinucleation, thickening of nuclear rim. However, there is enlargement of cytoplasm also, so that N/C ratio remains within normal limit. There may be perinuclear halo and leucocyte engulfment in the cytoplasm. In degenerated cells, nuclei vary in shape with irregular condensation of chromatin. But viable cells show round uniform nuclei.

2. Hyperplasia and regeneration -

Hyperplasia indicates increase in the number of cells. The individual cells show nuclear enlargment with regular outline and normal chromatin structures. There is only mild increase in the N/C ratio.

3. Metaplasia -

Metaplasia is the change in the type of adult cells in a tissue to a form which is not normal for that tissue. Metaplastic changes usually are of epithelial variety i.e. squamous metaplasia. They should be differentiated from malignant cells by the following criteria, (i) they remain in a flat sheet and contain adequate cytoplasm; (ii) chromatin arrangement in the nucleus is usually of benign nature.

O B S E R V A T I O N S

OBSERVATION AND RESULTS

A total numbers of 74 cases of oral lesions were examined during the study period. The age of the patients ranges from second to eighth decade of life. The youngest patient was of 17 years of age and oldest was of 73 years. Table I shows the age distribution of the 74 patients examined for study.

TABLE - I : Showing the age distribution in years of the 74 patients.

Age in years	Numbers of cases	Percentage
0 - 10	-	-
11 - 20	04	05.41
21 - 30	13	17.58
31 - 40	13	17.58
41 - 50	22	29.70
51 - 60	16	21.62
61 - 70	04	05.41
71 - 80	02	02.70
81 - above	-	-

Table I shows that the maximum number of cases 22 (29.7%) were in the age group 41-50 years and only 2 (2.7%) were in the group of 71-80 years of age.

Out of the total 74 cases, 60 were male and 14 were female giving a male-female ratio of 4.28 : 1. Table II shows the sex distribution of the patients.

TABLE - II : Showing the sex distribution

No. of cases	Male		Female		Male-Female ratio
	No.	%	No.	%	
74	60	81.08	14	18.92	4.28 : 1

TABLE - III : Showing religionwise distribution of the 74 cases examined.

Sl.No.	Religion	Number of cases	Percentage
1.	Hindu	68	91.90
2.	Muslim	06	08.10
3.	Christian	-	-
4.	Others	-	-

As shown in above table III, out of the total 74 cases examined, 68 (91.9%) were Hindus and 6 (8.1%) were Muslim.

TABLE - IV : Showing sex and relicionwise distribution of 74 patients examined.

Sl. No.	Sex		Hindu patients	Percentage	Muslim patients	Percentage
1-	Male	(60)	55	80.88%	05	83.33%
2-	Female	(14)	13	19.12%	01	16.67%
	Total		68		06	

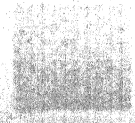
(Figures in parentheses indicates total No. of cases)

The table - IV shows that in both communities viz. Hindus and Muslims, Males predominated as compared to females.

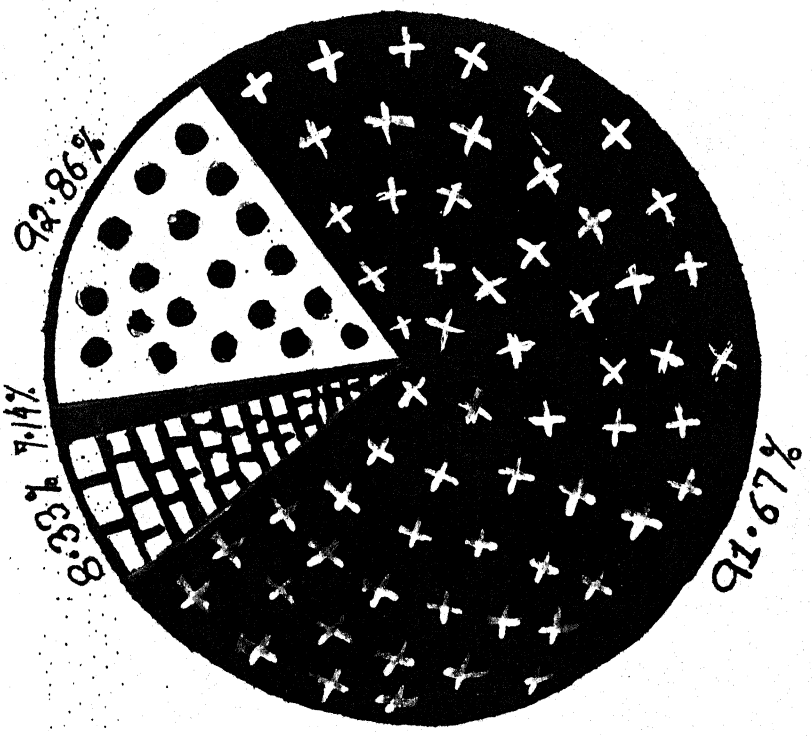
TABLE - V : Showing Socio-economic status of the patients

Total No. of cases	Poor	Middle	Rich
74	41 (55.4%)	31 (41.9%)	2 (2.7%)

Table - V shows distribution of 74 cases examined on the basis of Socio-economic status. 41 cases (55.4%) belong to poor class, 31 cases (41.9%) were in middle class group and only 2 cases (2.7%) belong to rich socio-economic status.



DISTRIBUTION OF PATIENTS ACCORDING TO SEX AND RELIGION.



HINDU (MALE)

HINDU (FEMALE)

MUSLIM (MALE)

MUSLIM (FEMALE)

TABLE - VI : Showing number and type of lesions examined by cytology.

S.No.	Classification	No. of lesions	Percentage of lesions
1.	MALIGNANT : (Squamous cell carcinoma)		
	- Well differentiated (Grade I)	11 (7)	14.87
	- Moderately differentiated (Grade II)	09 (6)	12.16
	- Poorly differentiated (Grade III)	03 (3)	4.06
2.	PRE-CANCEROUS :		
	- Leukoplakia without dysplastic changes	16 (3)	21.62
	- Leukoplakia with mild dysplastic changes.	16 (6)	21.62
	- Leukoplakia with moderate dysplastic changes	06 (3)	8.11
	- Leukoplakia with severe dysplastic changes	- -	-
	- Sub Mucous Fibrosis	02 -	2.70
3.	BENIGN :		
	- Oral Ulcers	09 (1)	12.16
	- Pemphigus vulgaris	02 (1)	2.70
	Total	74 (30)	

(Figures within parentheses show the number of cases in which biopsy was done).

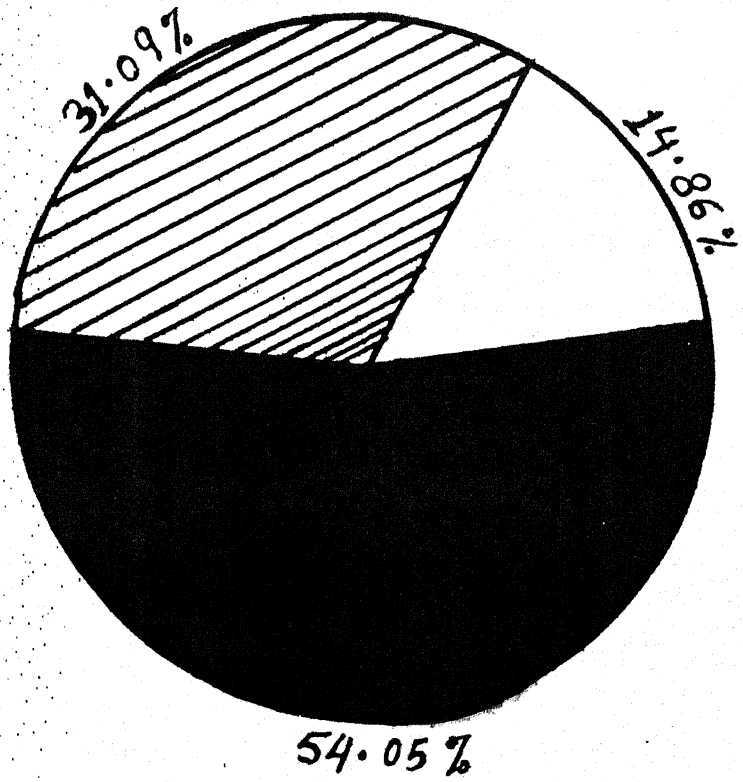
The table No. VI shows that out of total 74 cases examined cytologically, 23 cases (31.09%) were of Frank cancers, precancerous lesions 40 cases (54.05%) and 11 cases (14.86%) were benign lesions.

All the 23 cases of oral carcinoma were of epithelial origin. Out of 23 cancerous lesions, 11 cases (47.84%) were well differentiated (GradeI), 9 cases (39.12%) were moderately differentiated (Grade II) and 3 cases (13.04%) were poorly differentiated.

Out of 40 precancerous lesions, 38 cases (95%) were of leukoplakia and only 2 cases (5%) were of oral submucous fibrosis. Dysplastic changes were not observed in 16 cases (40%) of leukoplakia. Mild dysplastic changes were seen in 16 cases (40%) of leukoplakia and 6 cases(15%) of leukoplakia showed moderate degree of dysplastic changes.

Out of a total of 74 cases of oral lesions, there were 30 cases (40.54%) in which accompanying biopsy was done.

DISTRIBUTION OF CASES EXAMINED ACCORDING TO CANCEROUS, PRECANCEROUS AND BENIGN LESIONS



CANCEROUS

PRECANCEROUS

BENIGN

TABLE - VII : Showing age incidence in the cases of cancerous, pre-cancerous and benign oral lesions.

Age group	Oral cancer		Pre-cancerous lesions		Benign oral lesions	
	No.	%	No.	%	No.	%
0 - 10	-	-	-	-	-	-
11 - 20	-	-	3	7.50	1	9.10
21 - 30	3	13.04	8	20.00	2	18.20
31 - 40	4	17.40	8	20.00	1	9.10
41 - 50	6	26.08	11	27.50	5	45.44
51 - 60	5	21.74	9	22.50	2	18.20
61 - 70	3	13.04	1	2.50	-	-
71 - 80	2	8.70	-	-	-	-
81 - above	-	-	-	-	-	-
Total	23		40		11	

Table VII shows that most of the oral cancer cases 6 (26.08%) were observed in the 41-50 years age group, followed by 5 cases (21.74%) in group range from 51-60 years and lowest number of cases 2 (8.70%) were observed in 71-80 years age group. Minimum age was 30 years and maximum was 73 years.

Maximum number of the pre-cancerous lesions 11 (27.5%) were observed in 41-50 years age group, followed by 9 cases (22.5%) in 51-60 years age group. Only one case (2.5%) was observed in the 61-70 years age group. Minimum age was 18 years and maximum was 65 years.

TABLE - VIII : Showing sex incidence in the cases of cancerous, pre-cancerous and benign oral lesions.

S.No.	Lesions	Total No.of cases	Male		Female		Male Female ratio
			No.	%	No.	%	
1.	Oral cancer	23	19	82.60	04	17.40	4.75 : 1
2.	Precancerous lesions	40	34	85.00	06	15.00	5.66 : 1
3.	Benign oral lesions	11	07	63.64	04	36.36	1.75 : 1
	Total	74	60		14		

Table VIII shows that out of 23 cases of oral cancer, 19 (82.6%) were males and 4 (17.4%) were females. The male female ratio was 4.75 : 1.

Among 40 cases of precancerous oral lesions, 34 (85%) were males and 6 (15%) were females. The male female ratio was 5.66 : 1.

Out of 11 cases of benign oral lesions, 7 (63.64%) were males and 4 (36.36%) were females. The male female ratio was 1.75 : 1.

TABLE - IX : Showing relationship between type of lesions and different types of personal habits.

S.No.	Habit	Oral cancer		Precancerous lesions		Benign lesions		Total
		No.	%	No.	%	No.	%	
1.	Tobacco chewing either alone or with Pan Masala (Khaini, Gutka)	2	8.70	5	12.50	-	-	07
2.	Pan with tobacco chewing	-	-	2	5.00	-	-	02
3.	Sada Pan or Pan Masala without tobacco	-	-	1	2.50	-	-	01
4.	Smoking and tobacco chewing either alone or with pan	7	30.44	16	40.00	1	9.10	24
5.	Smoking alone	3	13.04	6	15.00	2	18.20	11
6.	Alcohol and smoking	3	13.04	3	7.50	-	-	06
7.	Alcohol, smoking and tobacco chewing	5	21.74	3	7.50	1	9.1	09
8.	Person without and addiction	3	13.04	4	10.00	7	63.60	14
	Total	23		40		11		74

Table IX shows that in Oral cancer, habit of smoking and tobacco chewing (either alone or with Pan) was found in 7 cases (30.44%). History of alcohol, smoking and tobacco chewing was available in 5 cases (21.74%), smoking alone in 3 cases (13.04%), alcohol and smoking in 3 cases (13.04%), and tobacco chewing (either alone or with Pan Masala) in 2 cases (8.7%) only. There were 3 cases (13.04%) in our study of oral cancer, which were without any addiction.

Smoking habit with tobacco chewing (either alone or with Pan) was found in 16 cases (40%) of Pre-cancerous lesions. Smoking alone was found in 6 cases (15%). Tobacco chewing (either alone or with Pan Masala) was available in 5 cases (12.5%). Alcohol and smoking in 3 cases (7.5%). Alcohol, smoking and tobacco chewing in 3 cases (7.5%). Pan with tobacco chewing in 2 cases (5%) and Pan chewing without tobacco in only one case (2.5%). 4 cases (10%) had no addiction (Table IX).

In Benign Oral lesions, Habit of smoking alone was found in 2 cases (18.2%), smoking and tobacco chewing (either alone or with pan) in one case (9.1%) and Alcohol, smoking and tobacco chewing in one case (9.1%). There were 7 cases (63.6%) which were without any addiction (Table IX).

DISTRIBUTION OF NUMBER OF CASES (74)
ACCORDING TO HABITS.

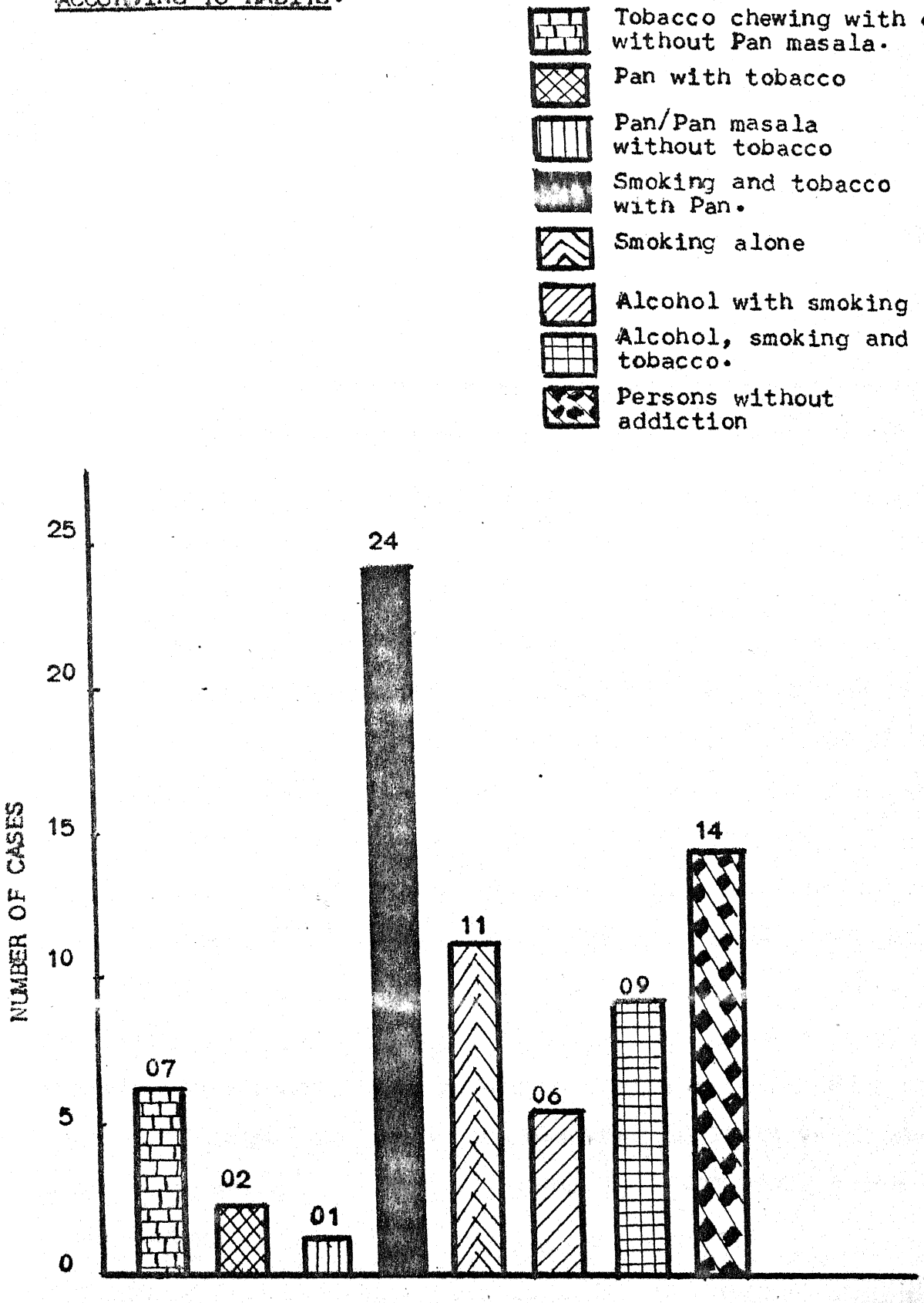


TABLE - X(a) : Showing relationship of cancerous lesions (20 cases) with duration of habits.

Total period of addiction (in years)	Tobacco chewing (Gutka)	Pan with tobacco chewing	Smoking and tobacco chewing with Pan	Smoking alone	Alcohol and smoking	Alcohol, smoking and tobacco chewing
0 - 10	-	-	1	-	-	-
11 - 20	-	-	2	-	-	1
21 - 30	2	-	2	1	-	2
31 - 40	-	-	1	1	1	-
41 - 50	-	-	1	1	2	2
Total	2	-	7	3	3	5

Table X(a) shows that 20 cases of oral cancer were addicted to various addictant for various range periods. 2 cases (10%) gave history of addiction to tobacco chewing for 21-30 years range period. 7 cases (35%) gave history of smoking and tobacco chewing with Pan. Out of these 7 cases, one case (14.29%) each was observed in 0-10 years, 31-40 years and 41-50 years range period of addiction where as 2 cases (28.58%) each were in 11-20 years and 21-30 years range period. 3 cases (15%) were smokers only which included 1 case (33.33%) each in 21-30 years, 31-40 years and 41-50 years range period. 3 cases (15%) were addicted for alcohol and smoking. Among these 3 cases, 1 case (33.33%) was observed in 31-40 years and 2 cases (66.66%) in 41-50 years. 5 cases (25%) gave history of addiction to alcohol, smoking and tobacco chewing. Out of these 5 cases, 2 cases (40%) each were observed in 21-30 years and 41-50 years while only one case (20%) in 11-20 years range period of addiction.

TABLE - X(b) : Showing relationship of Pre-cancerous lesions (36 cases) with duration of habits.

d of tion ears)	Tobacco chewing (Gutka)	Pan with tobacco chewing	Pan without tobacco	Smoking and tobacco chewing with Pan	Smoking alone	Alcohol and smoking	Alcohol, smoking and tobacco chewing
10	3	1	-	8	3	-	1
20	-	1	-	4	1	1	-
30	1	-	1	1	2	-	1
40	1	-	-	2	-	1	1
50	-	-	-	1	-	1	-
tal	5	2	1	16	6	3	3

Table X(b) shows that 36 cases of precancerous lesions gave ory of addiction to various addictants. 5 cases (13.88%) were co chewers only. Out of these 5 cases, 3 cases (60%) were addicted)-10 years range period and 1 case (20%) each was observed in 21-30 s and 31-40 years range period of addiction. 2 cases (5.55%) gave ory of Pan with tobacco chewing which included one case (50%) each -10 years and 11-20 years. Only one case (2.77%) was having habit an chewing without tobacco for 21-30 years range. 16 cases (44.32%) having habit of smoking and tobacco chewing with Pan. Out of these ases, 8 cases (50%) were observed in 0-10 years; 4 cases (25%) in 0 years; 1 case (6.25%) in 21-30 years; 2 cases (12.5%) in 31-40 s and 1 case (6.25%) in 41-50 years range period of addiction. 6 s (16.62%) were smokers. Out of these 6 cases, 3 cases (50%) were ing for 0-10 years; 1 case (16.66%) for 11-20 years and 2 cases ,24%) for 21-30 years range period. 3 cases (8.31%) were having it of accohol and smoking. Out of these 3 cases, 1 case (33.33%) was addicted for 11-20 years; 31-40 years and 41-50 years.3 cases

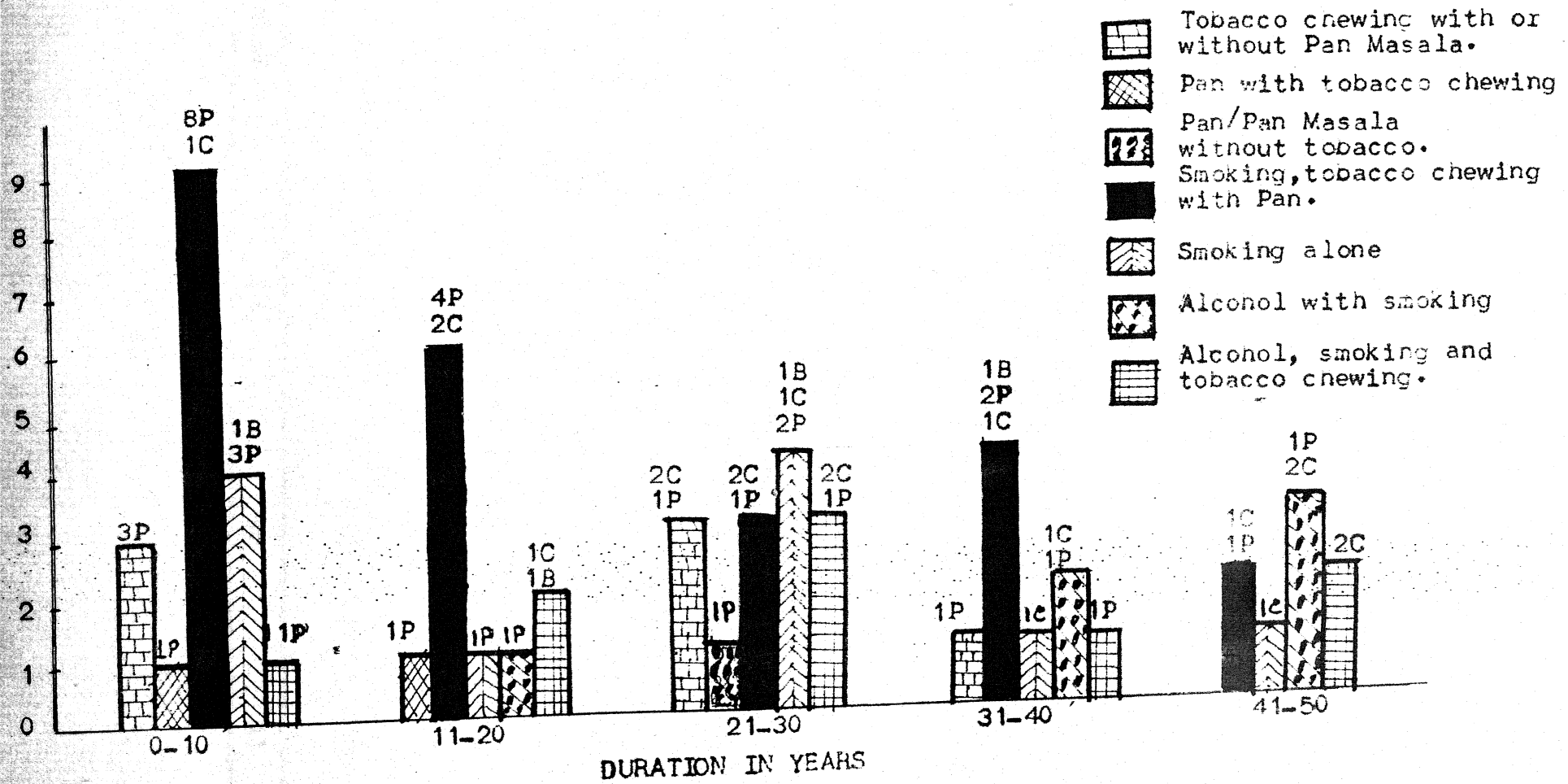
(8.31%) were addicted for alcohol, smoking and tobacco chewing. Out of these 3 cases, 1 case (33.33%) each was observed in 0-10 years, 21-30 years and 31-40 years range period.

TABLE - X(c) : Showing relationship of Benign lesions (4 cases) with duration of habits.

Total period of addiction (in years)	Tobacco chewing (Gutka)	Pan with tobacco chewing	Smoking and tobacco chewing with Pan	Smoking alone	Alcohol and smoking	Alcohol, smoking and tobacco chewing
0 - 10	-	-	-	1	-	-
11 - 20	-	-	-	-	-	1
21 - 30	-	-	-	1	-	-
31 - 40	-	-	1	-	-	-
41 - 50	-	-	-	-	-	-
Total	-	-	1	2	-	1

Table X(c) shows that only 4 Benign cases were addicted for various addictants. One case (25%) gave history of smoking and tobacco chewing with pan for 31-40 years range period. 2 cases (50%) were smokers. Out of these 2 cases, one case (50%) was smoking for 0-10 years and another one case (50%) for 21-30 years. One case (25%) was having habit of alcohol, smoking and tobacco chewing for 11-20 years range period of addiction.

DISTRIBUTION OF (60) CASES OF ORAL LESIONS ACCORDING TO DURATION WITH HABITS.



(P = Precancerous, C = Cancerous, B = Benign oral lesions)

TABLE - XI : Showing the sitewise distribution of the 74 cases.

Site	Oral cancer		Precancerous lesions		Benign lesions	
	No.	%	No.	%	No.	%
Lip	2	8.69	5	12.50	-	-
Cheek (Buccal mucosa)	7	30.43	29	72.50	3	27.27
Floor of mouth	-	-	-	-	-	-
Gingiva	1	4.36	1	2.50	-	-
Palate	-	-	1	2.50	-	-
Anterior 2/3 of the tongue	2	8.69	-	-	7	63.63
Posterior 1/3 of the tongue	2	8.69	-	-	-	-
Tonsillar region	9	39.14	-	-	-	-
Multiple sites	-	-	4	10.00	1	9.10
Total	23		40		11	

The table XI shows that in 9 (39.14%) out of 23 cases of oral cancer, tonsillar region was involved while 7 cases (30.43%) were of cheek (Buccal mucosa), 2 cases (8.69%) each of lip, anterior 2/3 of tongue and posterior 1/3 of the tongue. Only one case (4.36%) was of gingiva.

The cheek (Buccal mucosa) was the most common site of involvement with precancerous lesions 29 cases (72.5%). Next to the cheek involvement was the lip in which number of cases were 5 (12.5%). One case (2.5%) each was of gingiva and palate. In 4 cases there was involvement of more than one anatomical sites of oral cavity (Table XI).

Anterior 2/3 of the tongue was involved in 7 cases (63.63%) of benign lesions. 3 cases (27.27%) were of cheek (buccal mucosa) and 1 case (9.10%) was having involvement of lip, tongue and cheek simultaneously. (Table XI).

No case was observed from floor of mouth in our study.

SITE WISE DISTRIBUTION OF THE LESIONS (74 CASES).

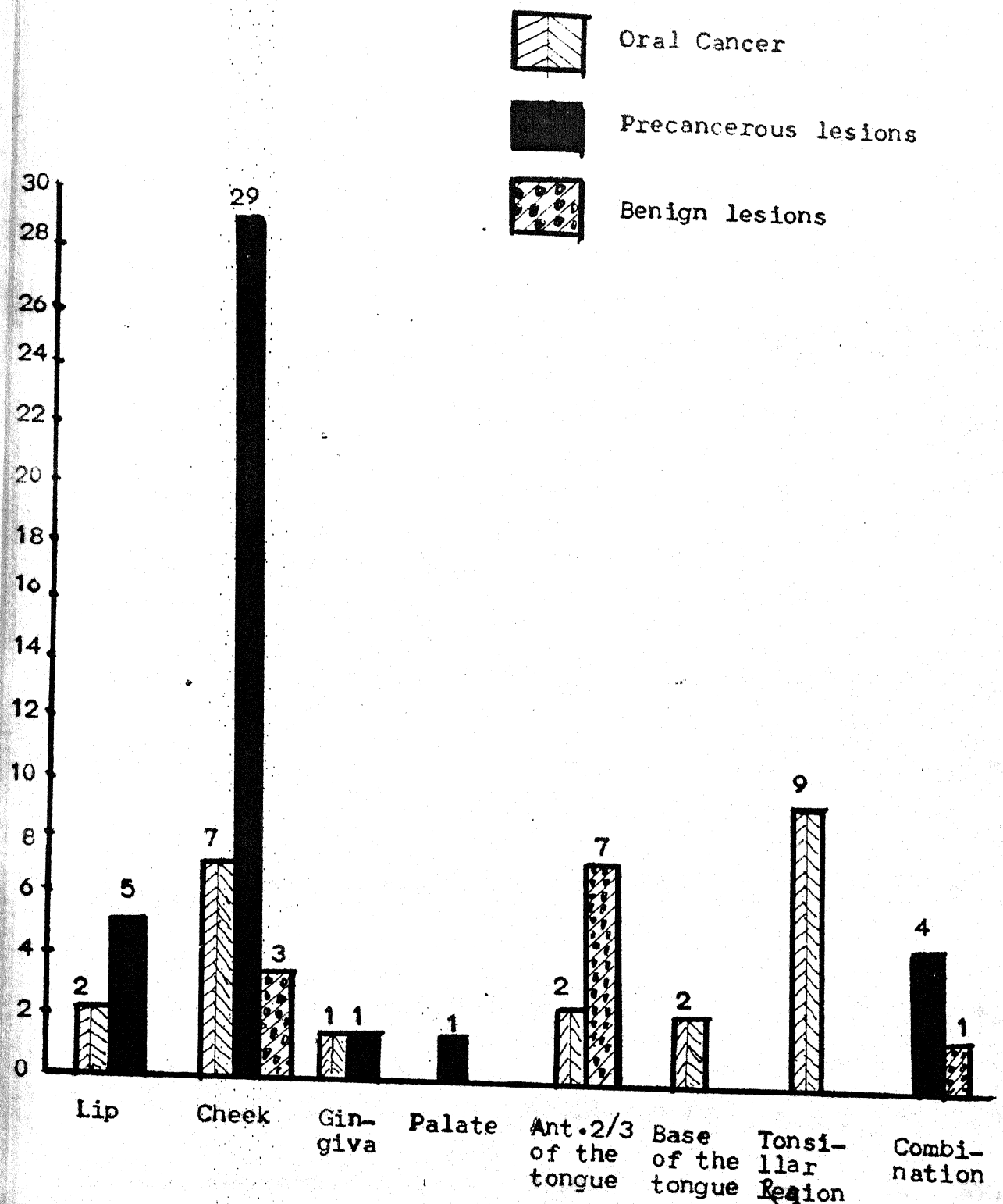


TABLE - XII : Showing the common clinical presentations of cancerous, precancerous and benign oral lesions.

Clinical features	Oral cancer		Precancerous lesions		Benign lesions	
	No.	%	No.	%	No.	%
White patch	-	-	36	90.00	1	9.10
Ulcer	7	30.43	9	22.50	10	91.00
Excessive salivation	12	52.17	-	-	-	-
Increased sensitivity to chillies and burning sensation	-	-	9	22.50	-	-
Trismus	4	17.39	4	10.00	-	-
Growth	16	69.56	-	-	-	-
Dysphagia	11	47.85	2	5.00	-	-
Hoarseness of voice	5	21.75	-	-	-	-
Otalgia	4	17.39	-	-	1	9.10
Pain and/or swelling in throat	10	43.50	-	-	-	-
Lymphadenopathy	8	34.80	-	-	2	18.20

Table XII shows that 16 cases (69.56%) out of 23 cases of oral cancer, sought medical advice for growth and 7 cases (30.43%) for non-healing ulcer in mouth. Excessive salivation in 12 cases (52.17%) and dysphagia in 11 cases (47.85%) were also frequent complaints. Other common clinical features were trismus in 4 cases (17.39%), hoarseness of voice in 5 cases (21.75%), otalgia in 4 cases (17.39%), pain and swelling in throat in 10 cases (43.5%) and lymphadenopathy in 8 cases (34.8%).

Out of 40 cases of precancerous lesions, 36 cases (90.0%) sought medical advice for white patches. 9 cases (22.5%) had complain of ulcer. 9 cases (22.5%) had intolerance to spicy food and complaint of burning sensation. 4 cases (10.0%) had complaint of trismus and only 2 cases (5.0%) of dysphagia (Table XII).

10 cases (91.0%) out of 11 benign lesions had complaint of oral ulcer. One case (9.1%) had complaint of otalgia and one case (9.1%) of white patch. 2 cases (18.2%) had lymphadenopathy (Table XII).

TABLE - XIII : Leukoplakia diagnosis revealed by cytology and confirmed by biopsy (12 cases).

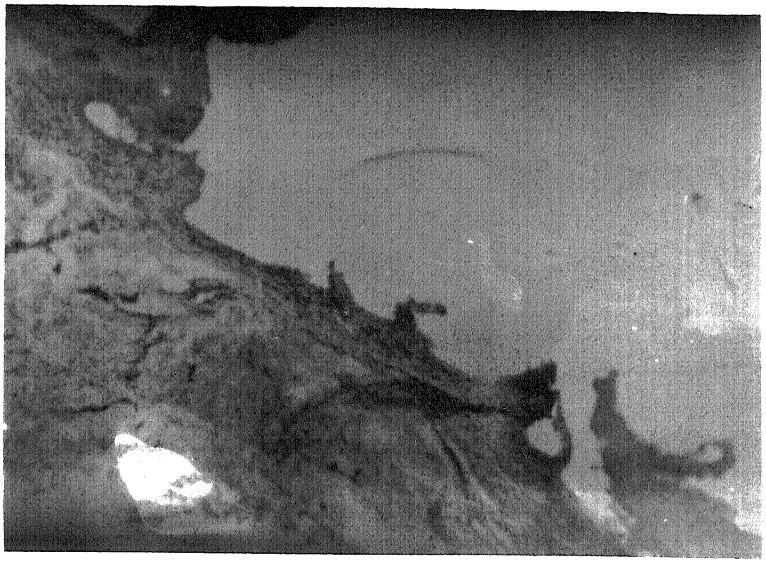
Cytology findings	Biopsy findings					Total
	Simple leukoplakia	Leukoplakia with dysplasia			No leukoplakia	
		Mild	Moderate	Severe		
Positive for leukoplakia	3 (25%)	3 (25%)	6 (50%)	-	-	12
Negative for leukoplakia	-	-	-	-	-	-

Table XIII shows that out of 38 cytologically diagnosed patients of oral leukoplakia, biopsy was done in 12 cases (31.58%), because other patients did not give their consent for biopsy. In these 12 cases, 3 cases (25%) had simple leukoplakia alone, 3 cases (25%) showed mild degree of dysplastic changes and in 6 cases (50%) moderate degree of dysplastic changes were observed. In no case cytology was negative which was to be confirmed by biopsy afterwards.

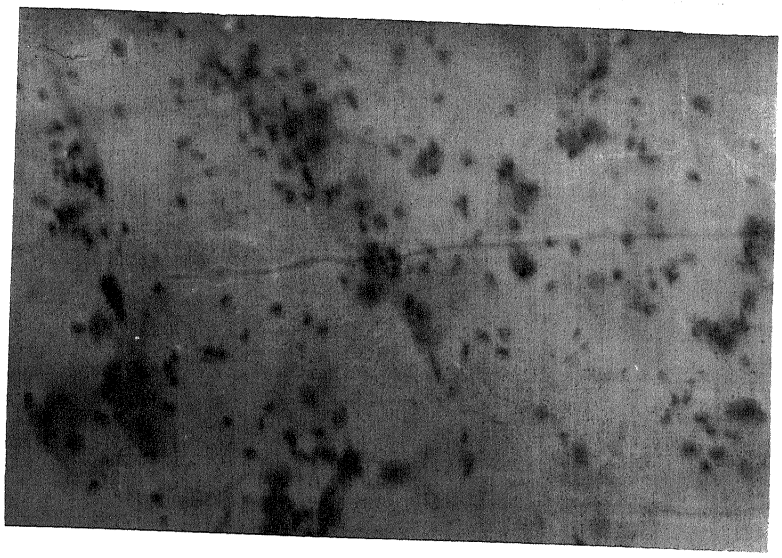
TABLE - XIV : Carcinoma diagnosis revealed by cytology and confirmed by biopsy (16 cases).

Cytology findings	Biopsy findings						Total
	Carcinoma in situ	Squamous cell carcinoma				No carcinoma	
		Grade I	Grade II	Grade III	Grade IV		
Positive for carcinoma	-	7 (43.75%)	6 (37.5%)	3 (18.75%)	-	-	16
Negative for carcinoma	-	-	-	-	-	-	-

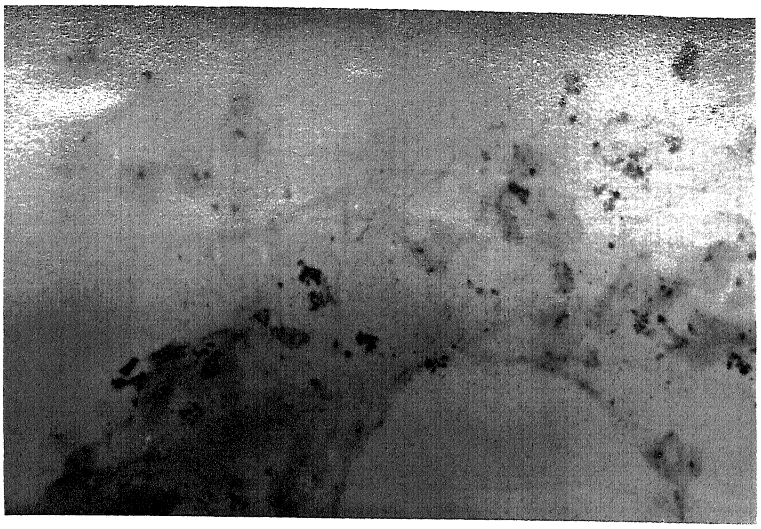
Table XIV shows that out of 23 cases of oral cancer, biopsy was done in 16 cases (69.56%). 7 (43.75%) out of 16 cases were well differentiated (Grade I) lesions. 6 cases (37.5%) showed grade II differentiation (moderately differentiated) and there were 3 cases (18.75%) with grade III differentiation (poorly differentiated). Carcinoma in situ case was not observed in present study. In no case cytology for carcinoma was negative which was to be confirmed by biopsy afterwards.



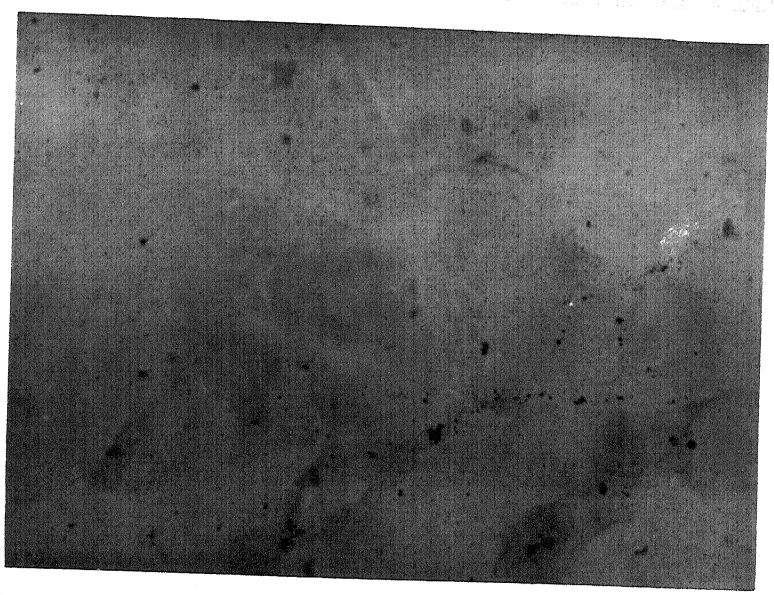
Histological section showing oral ulcer : denudation of covering epithelium along with inflammatory cells in the underlying tissues (H.E. stain, X 20).



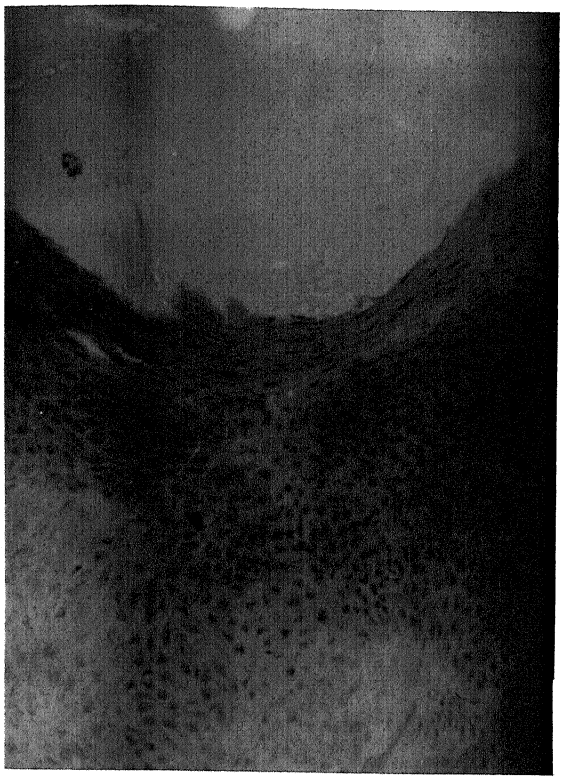
Oral cytology of Pemphigus vulgaris showing small squamous cells with markedly prominent nucleoli. (Papanicolaou's stain, X 80).



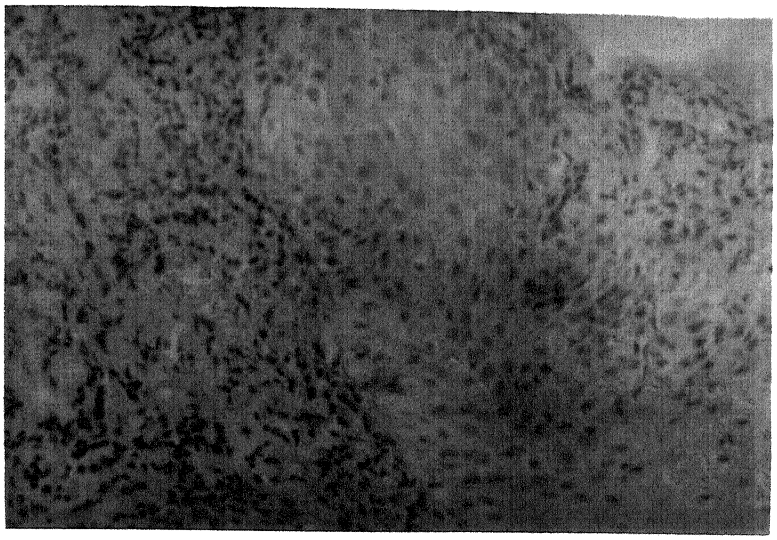
Oral cytology of leukoplikia showing superficial epithelial cells with perinuclear haloes.(Papanicolaou's stain, X 50).



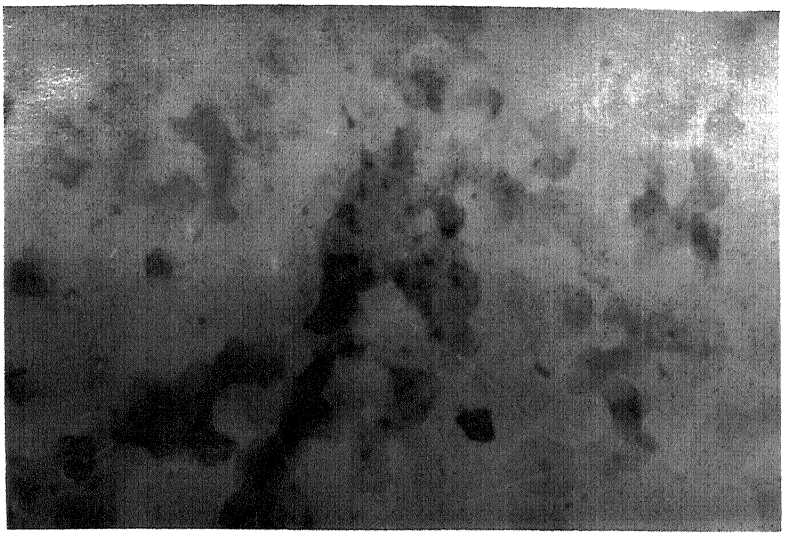
Oral cytology of leukoplakia showing completely keratinized anucleated epithelial cells.(Papanicolaou's stain, X 80).



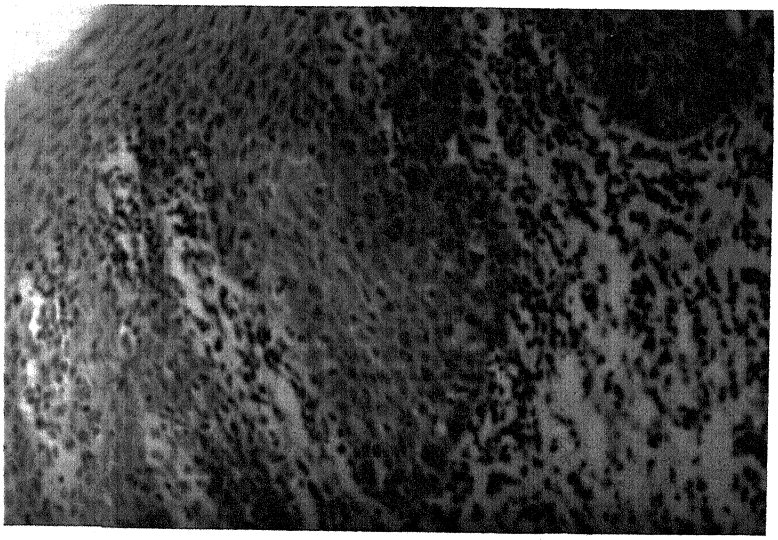
Histological section showing leukoplakia (H.E. stain X 50)



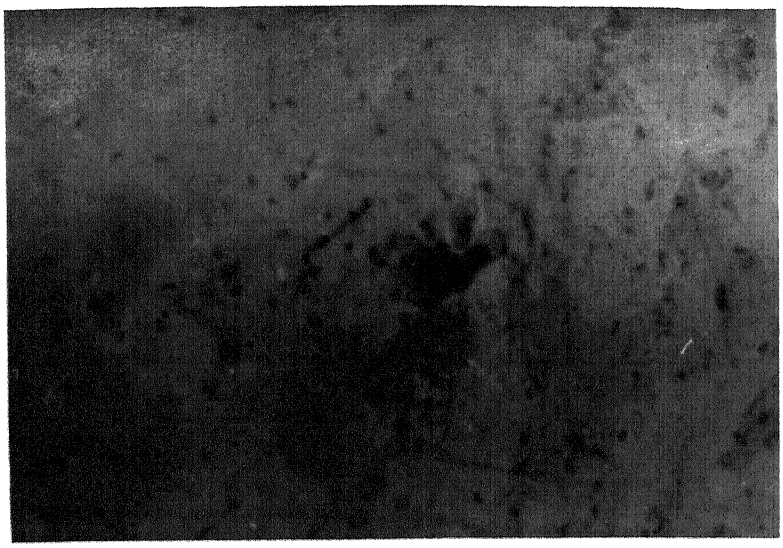
Histological section showing leukoplakia with mild dysplastic changes (H.E. stain, X 80).



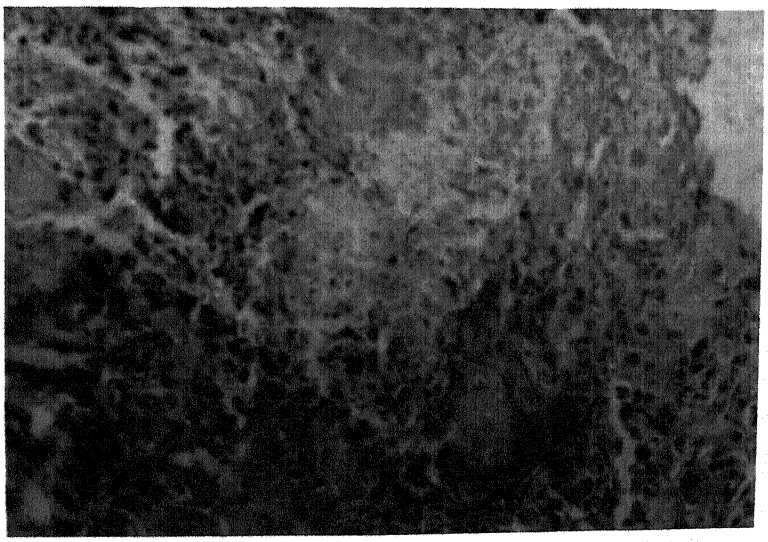
Oral cytology : Leukoplakia with moderate dysplastic changes (Papanicolaou's stain, X 50)



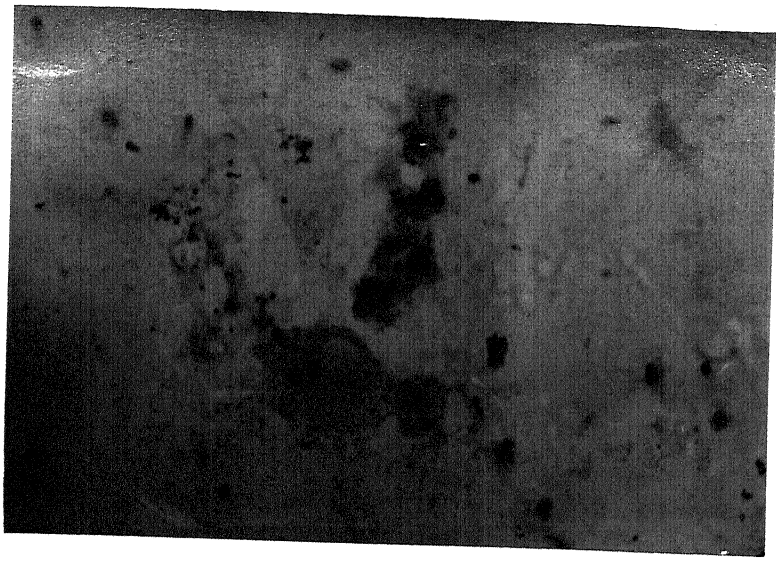
Histological section showing leukoplakia with moderate dysplastic changes (H.E. stain, X 80).



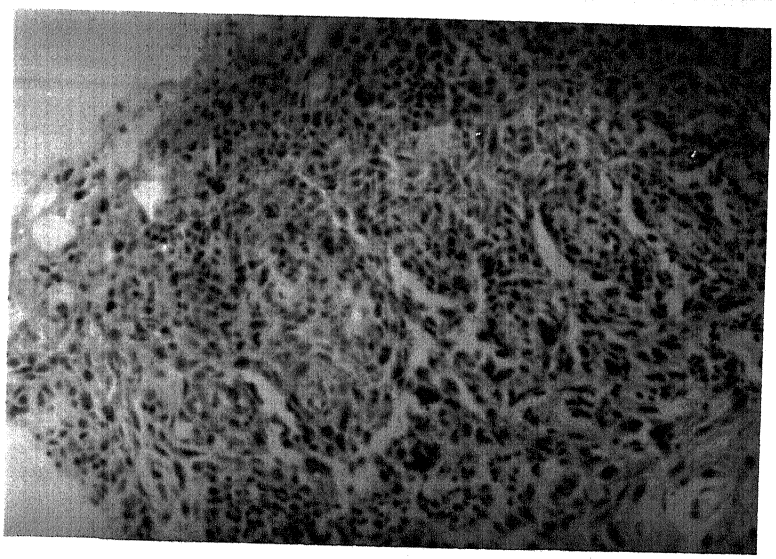
Oral cytology : Grade I epidermoid carcinoma with pleomorphic and hyperchromatic nuclei (Papanicolaou's stain, X 65).



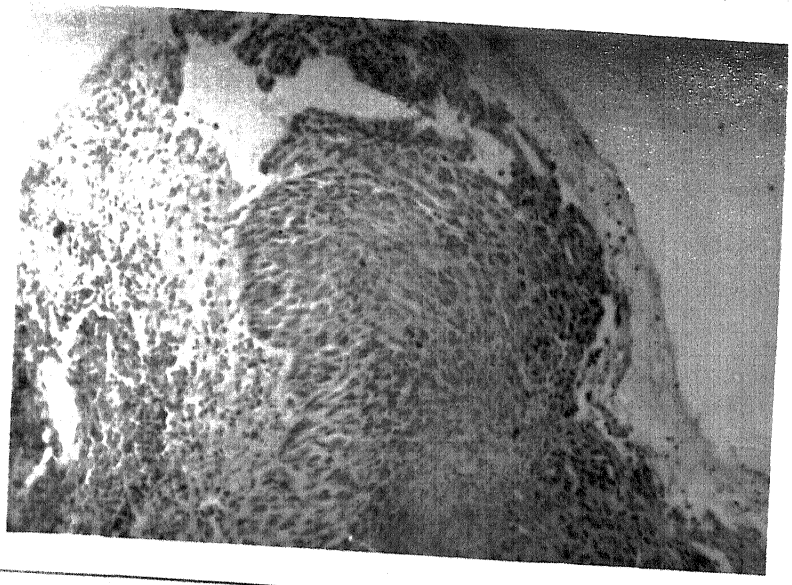
Epithelial pearl formation in epidermoid Grade I carcinoma (H.E. stain, X 50).



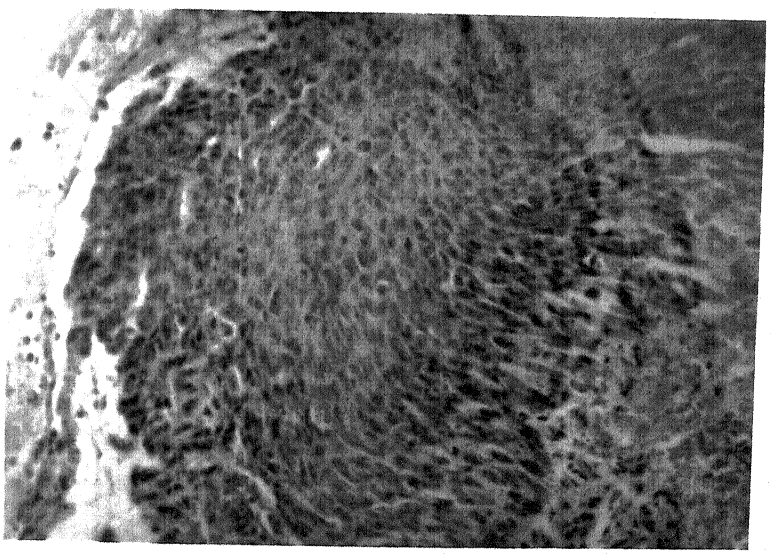
Oral Cytology : Grade II epidermoid carcinoma showing keratinized and nucleolar prominence (Papanicolaou's stain, X 100).



Histological section showing Grade II epidermoid carcinoma (H.E.stain, X 65).



Histological section showing epidermoid carcinoma Grade III (H.E.stain, X 50).



Histological section showing epidermoid carcinoma Grade III (H.E. stain, X 80).

D I S C U S S I O N

DISCUSSION

Asymptomatic abnormalities of the mouth are very common. Usually, their character is obvious, but some of these innocent - appearing lesions represent early cancer. The clinical diagnosis of mouth cancer at this early stage of development is completely unreliable and it is not practical to biopsy every minimal oral abnormality.

The successful application of exfoliative cytology to the detection of cancers at other sites suggested that cytology might be equally effective for the detection of early asymptomatic cancer of the mouth. Oral cytologic study has been recommended for the early diagnosis of carcinoma of the oral cavity (Montgomery, 1951; Hopp, E.S., 1958; Sandler et al, 1958; Silverman et al, 1958) and cases have been reported in which malignant cells were found in smears from innocent-appearing lesions, such as granulation tissue, ulcers, bleeding points and patches of leukoplakia, and from lesions which were not diagnosed by initial biopsy.

The cytodiagnosis of clinically suspected oral cancer is accurate. However, since it gives no information concerning the presence or extent of invasion, it should

not replace histilogic examination. Its role in such lesions is a supporting one. Despite the lower accuracy of cytologic diagnosis of residual or recurrent carcinoma, the practical value of this technique in the follow up of treated oral cancer is considerably greater, since repeated biopsies are not feasible; even if they were, it is doubtful that the precise biopsy site could be selected accurately, whereas oral smears permit the screening of the entire suspect area.

The present study was undertaken with a view to ascertain diagnostic accuracy of cytological study in all types of oral lesions and to correlate its diagnostic rate with that of histopathology.

In this present study a total number of 74 cases of different oral lesions were examined cytologically and also histologically where ever possible. All the cases were taken up from various out patient departments and admitted cases in the wards of Maharani Laxmi Bai Medical College, Jhansi.

In present study a maximum number of cases i.e. 22 (29.70%) were observed in 41-50 years age range (Table I) which was followed by 16 cases (21.62%) in the age range 51-60 years. There were only 2 cases (2.7%) beyond 70 years of age whereas the reported figures in literature (Fox,1925;

Prinz, 1928; Orr, 1930; King and Hamilton, 1931; Cumer, 1946; Cooke, 1956; Simpson, 1957 and Baruah, 1964) indicate maximum incidence in 5th and 6th decade of life. Average age reported by Fox (1925) was 48.4 years as compared to 41.75 years in present study. The average age was 41.5 years in Wahi et al (1961) and 32 years in Eichenlaub (1928). The earlier age incidence in the present series of cases may be explained on the basis of detection of lesion early and habit of smoking acquired at an early age in India.

Out of total 74 cases studied, there were 60 (81.08%) male and 14 (18.92%) female (Table II). The male-female ratio was 4.28 : 1. Preponderance of males in the present study is in agreement with the reported figures in the literature varying from 74 to 95 percent in males (Fox, 1925; Prinz, 1928; King and Hamilton, 1931; Cooke, 1956; Simpson, 1957; Wahi et al, 1961; Agarwal and Arora, 1964 and Samuel et al, 1969). This may be explained on the basis of low incidence of smoking habit in women. There were 68 (91.9%) Hindus and only 6 (8.1%) cases of Muslims community (Table III). In a similar study conducted by Wahi et al (1952, 1961) Hindus predominated over Muslims.

Mostly patients i.e. 41 cases (55.4%) belong to poor socio-economic status which was followed by 31 (41.9%) belonging to middle class. A higher incidence of precancerous

oral lesions in form of leukoplakia has been observed in persons of low socio-economic status (Wahi et al, 1961) and higher incidence of oral cancer in people of low socio-economic status was observed by Agarwal et al (1964) and Samuel et al (1969).

Out of total number of 74 cases examined by cytological means, there were 40 cases (54.05%) of pre-cancerous lesions i.e. leukoplakia and sub mucous fibrosis (2 cases) and a total of 23 cases (31.09%) of frank cancers. Benign lesions were 11 (14.86%) (Table VI).

In present study, out of the total 11 cases of benign oral lesions, 7 cases (63.64%) were male and 4 (36.36%) were female. Male-female ratio was 1.75 : 1. Maximum number of cases i.e. 5 (45.44%) were in the 41-50 years age group. Mostly cases i.e. 7 (63.6%) cases had no addiction to any addictants. 2 cases (18.2%) were smokers, 1 case (9.1%) was having habit of smoking and tobacco chewing with pan and habit of alcohol, smoking and tobacco chewing together was observed in one case (9.1%). Anterior 2/3 of the tongue was involved in 7 cases (63.63%), 3 cases (27.3%) were of cheek (buccal mucosa) and 1 case (9.1%) was having involvement of multiple site. Mostly (91%) benign lesions presented in form of oral ulcers and one case (9.1%) presented as white patches all over the tongue. Cytologically 9 cases (81.8%) were diagnosed as oral ulcer and 2 cases (18.2%) as oral manifestations of Pemphigus vulgaris.

Biopsy was done in only 2 cases of benign oral lesions which confirmed diagnosis of oral ulcer in one and pemphigus vulgaris in another case.

For the bulk of benign conditions that involve the mouth, cytology has not been reported of much diagnostic aid (Silverman et al, 1958).

Leukoplakia has been found to be commoner in Hindus as compared to Muslims or other communities. This is however expected considering the proportionate population. In the majority of cases the patients were not concious of the leukoplakic patches in the mouth. The symptoms were complained of mostly after the onset of induration or ulceration or in the form of increased sensitivity to chillies and burning sensation along with trismus. This is in accordance with various other workers (Bloodgood, 1921; Hansen and Eichenlaub, 1922; Fox, 1925; Prinz, 1928; and McCarthy, 1936). In the present study simple leukoplakia as well as leukoplakia with mild dysplastic changes were observed in approximately equal number (40%) whereas leukoplakia with moderate dysplastic changes was found in only 6 cases (15%). There was not a single case of leukoplakia with severe degree of dysplastic changes (Table VI).

Buccal mucosa (72.5%) was the commonest site of involvement which was followed by the lip (12.5%), gingiva

(2.5%), palate (2.5%) and other sites. This finding is in confirmity with that of Fox (1925), Cooke (1956) and Wahi et al (1961). In 4 cases (10%) leukoplakic patches were found at multiple site (Table XI).

It was concluded as a result of our observations that smoking and tobacco chewing either alone or with Pan play a major role (40%) in production of leukoplakic patches (Table IX). Other habits for example smoking alone, tobacco chewing alone, smoking along with alcohol and tobacco chewing were also supposed to play some role in production of leukoplakic patches in a lower number of cases. Thus from our study it is clear that both tobacco chewing and smoking have role in aetiology of leukoplakia. These findings are in agreement with findings of Cooke (1956). In our study 2.5% cases were Pan chewers without tobacco. It has been first remarked by Prinz (1928) that betel nut by liberating tannic acid irritate mucosa. This irritation leads to leukoplakia. Role of betel nut is also clear from our study.

A clear cut tendency was noted that the earlier the habit of tobacco chewing along with smoking started, the higher the risk of developing precancerous and also cancerous lesions (Table X(a) and X (b)). The maximum number of leukoplakia cases were observed in persons who were having the habit for upto 10 years followed by in group of 11-20 years.

Cases of oral carcinoma were observed more in Hindus and compared to Muslims or other communities. It is in accordance with findings of Wahi et al (1952) in whose study Hindus were 1.8 times more than Muslims. Our finding may be general reflection of population ratio of various religions.

In the present series of cases of oral cancer, maximum incidence was in 5th decade (Table VII). This is in accordance with Orr (1933), Kini and Rao (1937), Paymaster (1957), Wahi et al (1958), Baruah (1964), Agarwal and Arora (1964), Samuel et al (1969), Saran et al (1984) and Agarwal et al (1985).

Oral carcinoma was found to be more prevalent in males (82.6%) as compared to females (Table VIII). Male-female ratio was 4.75 : 1. This higher incidence in males is in accordance with Wahi et al (1958), Baruah (1964), Agarwal and Arora (1964), Jussawalla (1968, 1971) and Saran et al (1985).

Our observations revealed that smoking and tobacco chewing either alone or with pan play a major role in production of oral cancer (Table IX). This finding is in concurrent with certain other studies (Khanolkar, 1944, 1959; Sanghvi et al, 1955; Shanta and Krishnamurthi, 1963; Paymaster, 1971; Wahi, 1968; Reddy et al, 1975).

Habit of taking alcohol along with smoking and tobacco chewing was second major factor playing role in production of oral cancer. Wynder and Bross (1957) reported that alcohol had a direct influence and Jellinck and Jolliffe(1940) also reported that alcohol had an indirect influence on the development of cancer of the mouth. Smoking alone, alcohol and smoking, and tobacco chewing either alone or with Pan Masala were also supposed to play a significant role in production of oral carcinoma.

Most cases of carcinoma presented as oral growths (69.56%) and as non-healing ulcers (30.43%). Other presenting symptoms were excessive salivation (52.17%), Dysphagia (47.83%), Pain and swelling in throat (43.50%) and hoarseness of voice (21.73%) (Table XII). These findings are in accordance with Agarwal et al (1985).

The commonest site of oral cancer was Tonsillar region (39.14%) followed by buccal mucosa (30.43%)(Table XI). This finding is in accordance with Jussawalla (1980). Our findings are at variance with reports of Wahi et al (1965), Samuel (1969) and Gangadharan (1979) who had reported the cheek as the commonest site for oral carcinoma.

The risk of cancer lesion was found to be higher with increasing frequency of smoking, Pan and chewing tobacco in good quantity and for a longer duration. Most cases

of oral cancer (35%) were found to have smoking and tobacco chewing habit for 11-20 years and 21-30 years duration period (Table X-a). This statement is also supported by Wahi et al (1965).

Present study includes 23 cases of oral carcinoma, a incidence of 31.09%. Whereas the reported incidence of cancer of oral cavity has been 40% (Khanolkar, 1950), 35% to 40% (Agarwal et al, 1985) and 34.6% (Saran et al, 1984-85).

Out of 23 cases, 11 cases (47.84%) of carcinoma were well differentiated (Grade I); moderately differentiated (Grade II) were 39.12% and only 3 cases (13.04%) were of poorly differentiated (Grade III) variety. Carcinoma-in-situ cases were not observed. The highest incidence of grade I (well differentiated) carcinoma in our series is in accordance with the highest incidence observed by Krsus and PeresMesa (1966).

CORRELATION OF CYTOLOGY WITH HISTOPATHOLOGY :

It was found that both cytology and biopsy were almost equally reliable in so far as the diagnosis of oral lesions specially the cancer of mouth was concerned. Each method was subjected to some degree of error but their correlation was very high and statistically significant. In our seried out of 23 cases of oral cancer, biopsy could have been possible only in 16 cases i.e. about (69.56%).

TABLE - XV : Showing the results of cytodiagnosis in oral carcinoma of different investigators with the present series of cases.

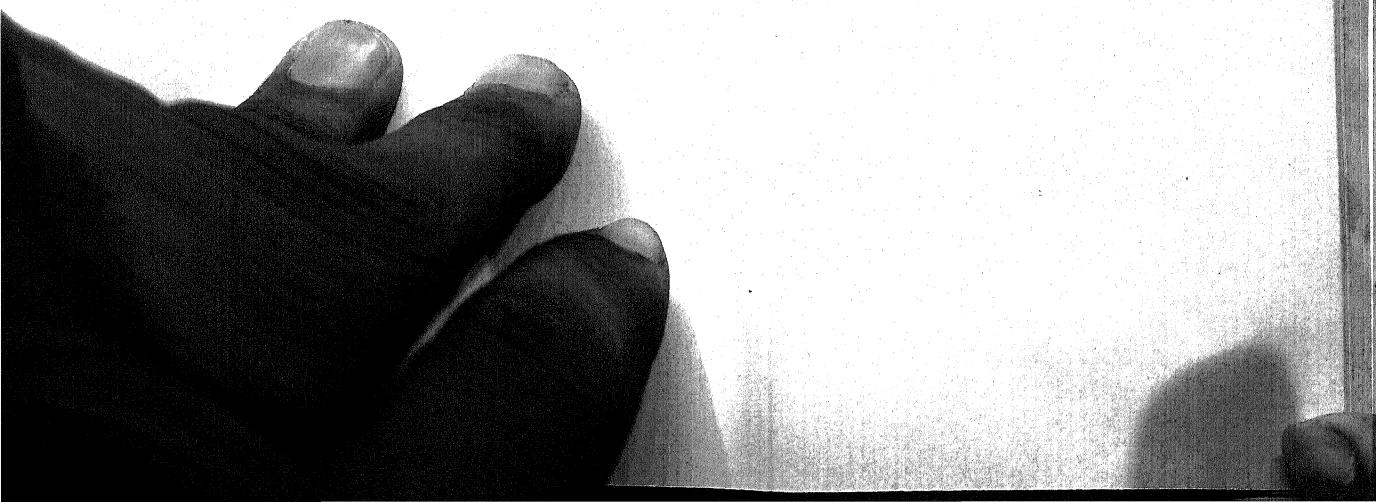
Investigators	Biopsy positive cases	Cytology positive cases	Diagnostic rate
Morrison et al (1949)	10	10	100%
Montgomery et al (1951)	15	13	86.7%
Wahi and Gupta (1954)	41	41	100%
Peters and Ryzinghani (1956)	194	186 (154)	96% (79%) excluding 'suspicious'
Hopp (1958)	39	38	97.2%
Silverman et al (1958)	18	18	100%
Cawson (1960)	31	25	81%
Ingram et al (1963)	17	14	82.3%
Selbach and Von Haam (1963)	93	90 (50)	96.8% (53.8%) excluding 'suspicious'
Sandler (1964)	315	307 (242)	97.4% (76.8%) excluding 'suspicious'
Gardner (1964)	18	18 (14)	100% (77.8%) excluding 'suspicious'
Present study	16	16	100%

In all these cases cytodiagnosis of carcinoma was well proved histologically thus giving an accuracy of over all hundred percent (Table XV). Here a point which is to be emphasized is that our cases have been purely selective one. Had this study been a survey of population, the percentage accuracy as a regard of diagnosis of carcinoma would have been fallen. Our results are in accordance with Wahi and Gupta (1954), Silverman et al (1958) who have reported 100% diagnostic rate. However mountgomery et al (1951) reported 66.7%, Peters and Ryasinghani (1956) 96% and Sandler (1962) has reported 82.14% diagnostic accuracy rate.

Carcinoma of oral cavity can be differentiated and graded successfully (Umiker, 1957; Gupta, 1968). In our study out of 16 cases biopsy proved carcinoma of oral cavity, there were 7 cases of Grade I (43.75%). It was followed by Grade II carcinoma (37.5%) and then Grade XXI carcinoma 3 cases (18.75%). There was not a single case of Grade IV carcinomas.

As regard leukoplakia in oral cavity, a positive cytological diagnosis can be certainly made which can be proved histologically. Both simple leukoplakia as well as leukoplakia with dysplastic changes can be successfully

differentiated and which can then be proved by histological means. In our series, out of 38 cases of oral leukoplakia, there were 12 cases biopsy proved (31.58%) leukoplakia. This giving an over all accuracy again 100%. This higher accuracy can be explained on the basis of cases taken selectively for the present study.



SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSION

Oral cancer is a significant health problem accounting for approximately 5 percent of all malignant tumours involving the body. "Death enters by mouth" is an old saying that is frequently true when speaking of infections or intoxications, but is especially applicable while considering the subject of oral malignancies and the lesions which are more liable to convert into malignancies in later stages, so called pre-malignant or pre-cancerous lesions like leukoplakia, melanoplakia, erythroplakia, sub mucous fibrosis, Lichen planus, stomatitis-nicotina palati, non healing ulcers etc. Either due to the ignorance or poor guidance to the sufferer the disease advances much and when the problem becomes social liability then patient runs up to consult the medical man but upto that time expectancy of life becomes less. Haldar, P.K. (1971) reported that effective treatment can be given to the patients in stages but early diagnosis is the crux of the problem. The fight against cancer can not be won only by improving the therapeutic techniques and the armamentarium but by catching the disease at an early stage.

The present study was conducted with a view to ascertain diagnostic accuracy of cytological study in all types of oral lesions and to correlate its diagnostic rate

with that of histopathology, having a look over the habits of the patients, distribution regarding age, sex, religion and site of the lesions etc.

Our observations have been discussed under the light of modern literature.

The following conclusions were drawn :-

The present study included 74 cases aged 11 years and above 70 years of age. There were 60 males and 14 females with a male-female ratio of 4.28 : 1. Mostly cases were Hindus (91.9%) and remaining were Muslims (8.1%).

Socio-economically 41 cases (55.4%) belong to poor status and 31 cases (41.9%) belong to middle class.

Out of 74 cases, there were 11 (14.86%) benign lesions, 40 cases (54.05%) precancerous lesions and oral cancers were 23 (31.09%).

Eleven cases of benign oral lesions included 7 males and 4 females. Mostly (63.63%) cases gave no history of addiction to any addictant. 2 cases (18.2%) gave history of smoking and one case (9.1%) had a habit of smoking, tobacco chewing with pan and alcohol intake. Site wise anterior 2/3 of tongue was mostly involved i.e. in 7 cases (63.63%), buccal mucosa in 3 cases (27.3%) and multiple site involvement in 1 case (9.1%). In 2 cases biopsies confirmed the cytological diagnosis thus giving

an overall accuracy of 100% as regard benign lesions. For the bulk of benign conditions that involve the mouth, cytology has not reported of much diagnostic aid (Silverman et al, 1958).

Out of 40 precancerous lesions, 38 cases (95%) were of leukoplakia and only 2 cases (5%) of oral submucous-fibrosis. Simple leukoplakia as well as leukoplakia with mild dysplastic changes were obserbed in approximately equal number (40%) whereas 6 cases (15%) of leukoplakia showed moderate degree of dysplastic changes.

Leukoplakia was found to be higher in the age range 41-50 year group.

Most of the leukoplakia cases (90%) presented clinically as white patches and buccal mucosa (72.5%) was the commonest site of involvement followed by lip (12.5%).

Habit of smoking and tobacco chewing either alone or with pan play a major role (40%) in production of leukoplakic patches.

Mostly oral carcinomas were of epithelial origin (100%). Out of 23 cancerous lesions, 11 cases (47.84%) were well differentiated (Grade I), 9 cases (39.12%) were moderately differentiated (Grade II), 3 cases (13.04%) were poorly differentiated.

Maximum number of oral carcinoma were observed in age group 41-50 year range.

Habit of smoking and tobacco chewing either alone or with pan play a major role (30.44%) in production of oral cancer. Habit of taking alcohol along with smoking and tobacco chewing was second major (21.74%) factor in causation of oral carcinoma.

Mostly carcinoma presented as oral growths (69.56%) and as non-healing ulcers (30.43%) in some cases.

Site wise mostly cases of oral carcinoma were of tonsillar region (39.14%) followed by buccal mucosa (30.43%) involvement.

The risk of cancer was found to be higher with increasing frequency of smoking, Pan and chewing tobacco in good quantity and for a longer duration. Most cases of oral carcinoma (35%) were found to have smoking and tobacco chewing habit for 11-20 year and 21-30 years duration period.

It was concluded from our study that the earlier the habit of tobacco chewing along with smoking started, the higher the risk of developing pre-cancerous and cancerous lesions.

It was found that both cytology and biopsy were almost equally reliable in so far as the diagnosis of oral

lesions. As regard leukoplakia in oral cavity, out of 38 cases, biopsies were performed in 12 cases which confirmed the cytological diagnosis giving an overall diagnostic accuracy of 100%.

Simple leukoplakia can be very easily differentiated from leukoplakia with dysplastic changes by cytology.

Oral carcinomas are very easily diagnosed by cytology smears. So much so that they can be graded accordingly.

There were 16 histologically proved cases of oral carcinomas out of 23 cases. The 100% diagnostic accuracy was acheived.

Cytodiagnosis of oral cancers is a simple, bloodless and rapid diagnostic method. Collection of material with wooden spatula is quite convenient and trustworthy.

Oral cytology is an excellent adjunct to follow up studies of treated and untreated oral cancers and may be an effective tool in the early detection of cancer and precancerous lesions of the mouth.

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APPENDIX

WORKING PROFORMA

CYTODIAGNOSIS OF ORAL LESIONS WITH HISTOPATHOLOGICAL CORRELATIO

Sl.No._____

CLINICAL DATA

1. Patient's Name _____ MRD/OPD No._____
2. Address _____ Age/Sex _____
   _____ Ward/Bed No._____
3. Occupation _____
4. Physician/Surgeon Incharge Dr. _____
5. Chief complaints : 1-
   2-
   3-
6. Habits (Personal history) : 1- Alcohol 2- Smoking
   3- Pan/Betal nut 4- Pan Masala
   5- 6-

LOCAL/GENERAL EXAMINATION

SYSTEMIC EXAMINATION

PATHOLOGICAL DATA

Cytopathology No._____

Histopathology No._____

Cyto-histopathology correlation